STUDIES ON THE OXYGENATION OF THE FOETUS IN NORMAL AND ABNORMAL PREGNANCY

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INTRODUCTION

For at least forty years pathological and functional changes in the placenta have been shown to be closely linked with the toxaemic states of pregnancy. In 1914, Young[1] showed an associated placental infarction probably due to ischaemia. Since then a considerable number of investigations correlating pre-eclampsia and the placenta have been carried out.

Among others, Tenney[2,3] and Hughes et al[4] have shown degenerative histological changes in the placentae of toxaemic pregnancies.

In 1939, Page[5] re-emphasised the association between toxaemia and placental ischaemia and since then he and many other investigators[6-13] have added weight to the theory that toxaemia is caused by, or related to, impairment of the placental blood supply.

Although placental ischaemia and dysfunction have been shown and it has long been known that babies born of toxaemic mothers are smaller than normal (Ludlow[14], Dieckmann[15]) and also show a much higher foetal mortality[16-22] which includes a large percentage of cases of intra-uterine death, very little investigation of the oxygen transfer of the placenta has been carried out.

Since 1930 (Eastman[23]) several investigations of the oxygen levels in the umbilical cord blood at birth

have been carried out. (Eastman, 1932[24], 1936[25]; Haselhorst and Stromberger, 1930-32[26-28]; Smith, 1939[29]; Dieckmann and Kramer, 1944[30]; Watts et al, 1951[31]). These have mainly been concerned with factors initiating respiration in the foetus and the effects on the oxygen levels caused by administering oxygen and anaesthetic gases to the mother in labour.

Only two investigations (Clemetson and Churchman, 1953[32], Walker and Turnbull, 1953[33]) have been directed specifically towards the effects of pre-eclampsia on the cord oxygen levels at birth and the results of these showed a marked variation. The former suggested a normal oxygenation of the foetal cord blood in the placenta while Walker showed an arterial saturation in the cord equivalent to about one half of that found in normal cases. In both instances relatively few cases were investigated and it was considered to be of advantage to try to make a further study of as many cases of pre-eclampsia as possible for comparison with normal pregnancy.

It was essential to compare the cases with due regard to the duration of pregnancy as Barcroft (1940 - sheep[34]) and Walker (1953 - human[33]) had shown a variation in the oxygen levels of cord blood which was dependent on the maturity alone, and it was decided to

extend the investigation to include the study of the effect, if any, of maturity in cases of normal pregnancy.

This may be of considerable value as the question of postmaturity has long been the subject of argument. Among others, Calkins[35], Latto[36], Hill[37], Lartz[38] and Willson[39] maintain that there is no danger to the foetus from prolongation of pregnancy without some additional complication such as toxaemia or disproportion, while others (Rathbun[40], McKiddie[41], Hamilton[42], Racker[43], Clifford[44] and Walker[45]) show an increase in foetal mortality, with an increase in the number of unexplained deaths, in cases where the pregnancy is unduly prolonged. Some of the latter consider any pregnancy of over 287 days to be postmature while others give a wider margin. Although several authors showed an increased difficulty in delivery due to increased size of the foetus, disordered uterine action, etc., this alone did not account for the total number of foetal losses and, once more, placental insufficiency was suggested as the cause of death.

This investigation is therefore directed towards finding out whether or not oxygenation of the foetal cord blood is impaired in infants delivered (a) postmaturely and (b) from mothers suffering from toxaemia.

As there is no method of studying the foetal circulation "in utero" a comparative study at birth has been made with conditions kept similar as far as was possible. The main difficulty encountered was in obtaining a sufficient number of comparable cases of toxaemia as the majority had instrumental or operative deliveries. Several cord bloods from such deliveries were also included in the study although a considerable number of variable factors influenced the results obtained.

The main study was made on the oxygen and carbon dioxide contents of the cord bloods obtained after uncomplicated spontaneous vertex deliveries where there was no evidence of cord obstruction and where chloroform was administered only with the birth of the head as is routine treatment in the Simpson Maternity Pavilion.

METHODS AND MATERIAL

All the cases were delivered in the Simpson Memorial Maternity Pavilion, and most were attended personally.

In all, 240 cord bloods have been studied. The majority were obtained after spontaneous vertex deliveries, where chloroform was administered with the birth of the head. This gave a basis for the comparison of normal term, premature, postmature, pre-eclamptic and hypertensive groups. Although the results obtained may not give a true picture of the oxygen levels in the umbilical cord "in utero", the conditions of the experiment were as nearly constant as possible.

Other cases included four twin pregnancies, a few breech deliveries, fifteen forceps deliveries and a series delivered by Caesarean section.

Immediately after delivery, and before the onset of respiration, the cord was occluded simultaneously at both ends to stop all circulation and four pairs of Spencer Wells forceps were applied - two at each end. A variable length of cord - usually about ten inches - was obtained by cutting between the forceps. The blood from the umbilical vein and arteries was immediately aspirated under liquid paraffin, transferred anaerobically to heparinised containers and

analysed at once.

Oxygen and carbon dioxide contents were estimated in duplicate by the method of Van Slyke and Neill (1924)[46] modified as described by Orcutt and Waters (1937)[47] in the constant-volume manometric apparatus, samples of 0.5 ml. of blood being used for each determination. Duplicate results agreed to within 0.06 vols. per cent. All the estimations were done either personally or under direct supervision.

Oxygen capacities were estimated in duplicate, also by the Van Slyke method, where sufficient blood was available. Most samples from the umbilical vein were sufficient for capacity determinations - about 5 - 10 ml. being obtained - but in almost half of the samples from the arteries there was insufficient, only about 2 - 3 mls. being obtained.

Haemoglobin levels were estimated in duplicate on both of the samples by the Spekker photo-electric absorptiometer - measuring oxyhaemoglobin after adding a known volume of blood (0.05 ml.) to 10 mls. of 0.007N ammonia solution (Arnold, 1949[48]). Duplicate readings agreed within 0.1 Gm.%.

Occasionally there was some discrepancy between the levels of haemoglobin in the vein and arteries, probably due to some degree of stasis caused by

kinking of the blood vessels. This was confirmed by estimations of the oxygen capacity of both samples where possible. Since oxygen capacities were not estimated on each sample, estimations were used to check the haemoglobin levels, confirming that 1 Gm. of haemoglobin (foetal and adult types) combines with 1.34 mls. oxygen. For consistency of results the oxygen saturation per cent was calculated from the haemoglobin levels in each sample.

Records were kept of the weights of the infants and the placentae. In abnormal cases records were kept of placentae showing macroscopic infarction.

Figures are given for the coefficient of oxygen utilisation which is a method of recording the amount of available oxygen used by the foetus.

$$\text{Coefficient of } O_2 \text{ utilisation} = \frac{(O_2 \text{ content (vols.\%) Umb. vein}) - (O_2 \text{ cont. Umb. arts.})}{O_2 \text{ content Umb. vein}}$$

The figures give an indication of the oxygen reserve in the blood returning to the foetus, reserve becoming lower as the coefficient approaches 1.00.

For all statistical comparisons the following

methods were used.[49]

Standard Deviation (s)

n = no. of cases in the group

x = individual results

$\bar{x}$ = mean result of the group

$$\therefore s = \pm\sqrt{\frac{S(x-\bar{x})^2}{(n-1)}}$$

Standard Error of the Mean (e) = $\pm \frac{s}{\sqrt{n}}$

Standard Error of difference between the means ($\bar{a}$ & $\bar{b}$)

$$e(\bar{a} - \bar{b}) = \pm\sqrt{e\bar{a}^{-2} + e\bar{b}^{-2}}$$

Statistical factor (t)

$$t = \frac{\text{Difference between the two means}}{\text{Standard error of difference between the means}}$$

$$= \pm \frac{\bar{a} - \bar{b}}{e(\bar{a} - \bar{b})}$$

P - Probability of chance variation

Obtained from tables by the result of t and the

no. of cases

$P = 0.10$ - probably significant

$P \leqslant 0.05$ - significant

$P \leqslant 0.01$ - highly significant

RESULTS OF PREVIOUS INVESTIGATIONS

Before giving the results obtained in the present study it would be of advantage to review some of those obtained by previous authors. As has been mentioned, these investigations have been largely directed towards the effects on the oxygen levels at birth of various types of anaesthesia, and several methods of delivery have been studied.

As far as can be deduced the cases studied were unselected - no indication is given of the normality or duration of pregnancy.

Table I shows some of the average results of previous studies with special reference to the type of anaesthesia and the method of delivery. The haemoglobin levels were calculated from the oxygen capacities.

There is a wide range of results in the per cent oxygen saturation in both the vein and the arteries. In the cases of spontaneous vertex deliveries in which no anaesthetic, or ether, or chloroform, was administered the oxygen saturation in the umbilical vein varied from 50% to 66%. In the spontaneous vertex deliveries where large quantities of nitrous oxide were administered the oxygen saturation of the blood reaching the foetus was considerably lower, e.g. 32.6%.

The oxygen saturation in the cord vessels of

TABLE I

AUTHOR	TIME OF SAMPLING	NO. OF CASES	DELIVERY	ANAESTHETIC	OXYGEN SATURATION %		Hb. GMS%
					UMB. VEIN	UMB. ARTS.	
HASELHORST & STROMBERGER (1931)[27]	Birth	4	Caes. Sect	Local or Spinal	41-62	-	
EASTMAN (1930)[23]	In utero	1	Caes. Sect		63.6	30.1	
	Birth	15	Spont.Vert.	Nil	50.5	15.9	15.5
EASTMAN (1936)[25]	Birth	4	2 Vert, 2 F.D.	Chloroform	50.0	< 20	
		8	Low Forc.	Ether	45.0	-	
		11	Spont.Vert.	Nitrous Ox. 80%	41.3	22.5	
SMITH (1939)[29]	Birth	21	Spont.Vert.	Ether	57.1	15.8	15.9
	Birth	20	Spont.Vert.	Nitrous Ox.	32.6	9.9	
	Birth	19	Mixed	Cycloprop.	44.0	17.0	
DIECKMANN & KRAMER (1944)[30]	In utero	3	Caes. Sect.	Spinal	44.0	12.4	14.4
	In utero	Serial Samples	Caes. Sect.	Spinal + oxygen	62.0	14.2	
WATTS ET AL (1951)[31]	Birth	8	Spont.Vert.	Ether	66.0	-	15.4
	Birth	17	Low Forc.	Ether	68.0	-	
	Birth	32	Mixed	Spinal	62.0	-	
	Birth	13	Mixed	Caudal	60.0	-	
	Birth	8	Mixed	Spinal (Drop in BP)	41.0	-	

infants delivered by Caesarean section was also very varied. Eastman[23] suggested that the saturation was higher in cases delivered by section but Haselhorst and Stromberger[28] thought that vaginal delivery raised the level of oxygen saturation.

It is interesting to note the increase in oxygen saturation shown by Dieckmann and Kramer[30], who took serial samples at intervals of up to 15 minutes from a loop of cord exposed at section, before and after the administration of oxygen to the mother. In three cases the oxygen saturation in the vein was raised by 12.3%, 32.0%, and 9.7%.

Haemoglobin levels in the cord at birth have also been the subject of many investigations and a wide range of results have been obtained. For example:-

1) Mugrage & Andresen[50](1936)	40 cases	17.1 Gms.% (13-20)
2) Guest et al[51](1938)	34 cases	17.9 Gms.% (13-22)
3) Mollison[52](1951)	134 cases	16.6± 1.5 Gms.%
4) Walker[33](1953)	145 cases	16.5 (15 - 18.6) at term
5) Marks et al[53](1955)	221 cases	16.9 ± 1.62 (12.3 - 22.0)

Marks[53] did not show any change in the haemoglobin level with increasing maturity or toxaemia, as did Walker[33]. His group of 221 cases were 30 - 44 weeks pregnant and the highest levels recorded were not accounted for in any way by postmaturity or toxaemia.

TABLE II.

AUTHOR	PREGNANCY	DURATION OF PREGNANCY	NO. OF CASES	DELIVERY	ANAESTHETIC	OXYGEN SATURATION %		Hb. GMS %	OXYGEN CAPACITY
						UMB. VEIN	UMB. ARTS.		
CLEMETSON & CHURCHMAN,[32] (1953)	Normal	Term	15	Sp.Vert.	½% Trilene	63.2	31.5	15.9	21.3
	Cooley's Anaemia	"	1	"	"	35.4	2.0	18.4	24.6
	Exclud. above	"	14	"	"	65.7	34.8	15.7	21.1
	Cord round neck	"	9	"	"	62.3	5.9	16.3	21.8
	Short Cord	"	3	"	"	63.5	4.2	15.4	20.7
	Toxaemic	(6 - 40) (1 - 36)	7	"	"	62.3	16.1	17.2	23.0
	Normal	Term	4	L/S Caes.	Spinal + O_2	43.1	10.8	14.1	18.9
	Toxaemic	"	4	L/S Caes.	Cyclo. + O_2	14.9	5.0	15.5	20.8
WALKER & TURNBULL,[33] (1953)	Normal	21-30	6	Caes.S	Spinal	69.4	34.3	14.3	19.1
	"	39-40	6	"	"	56.6	23.7	15.4	20.7
	"	42-43	6	"	"	34.3	6.8	18.3	24.5
	Toxaemic	34-39	5	"	"	23.7	7.4	17.8	23.8
	"	37	1	Sp.Vert.	Nil	46.8	20.8	16.6	22.2
WALKER,[45] (1954)	Normal	38	1	Sp.Vert.	Nil	50.0	2.4	17.8	23.8
	"	39-41	7	"	Nil	53.7	22.0	16.3	21.8
	"	42-43	2	"	Nil	28.7	9.95	17.7	23.7

A separate Table - II - shows the average results obtained by Clemetson and Churchman (1953)[32], and Walker and Turnbull (1953[33] and 1954[45]) whose researches were specifically directed towards the effect of pre-eclampsia on the oxygen levels in the vessels of the umbilical cord. It should be mentioned here that, whereas Clemetson used the Van Slyke and Neill method of estimating the oxygen content of the blood samples, Walker used the technique of Roughton and Scholander (1943)[54].

Clemetson's cases were both spontaneous vertex and Caesarean section deliveries but no particular reference was made to the duration of the pregnancy or of the toxaemia apart from one case of pre-eclampsia delivered at the 36th week. The normal cases (15) showed an average oxygen content in the umbilical vein of 13.1 vols. % (8.7 - 15.8) giving the percentage oxygen saturation of 63.2%, and in the arteries 6.6 vols. % (0.5 - 10.6) giving a saturation of 31.5%. When one case of Cooley's anaemia (U. vein - 35.4%, U. arteries - 2.0%) is excluded the average results are:- Umbilical vein 65.7% (49.7 - 83.6); umbilical arteries 34.8% (23.0 - 50.2) with an arterio-venous difference of about 31%. Even in this relatively small group the range of results was markedly scattered.

The average oxygen capacity was 21.1 volumes per cent which is equivalent to a haemoglobin level of 15.7 Gms. per cent with a range of 13.8 - 17.8 Gms.

In 9 cases in which the cord was obstructed, or potentially so, by being round the neck of the foetus the oxygen saturation was found to be:- Umbilical vein - 62.3% (40.5 - 80.3); Umbilical arteries - 5.9% (0.8 - 11.1). Normally saturated blood was apparently reaching the foetus but more oxygen was being removed by the foetus, probably due to a slowing of the circulation rate, and the blood returning to the placenta showed a much lower level of saturation than in a non-obstructed case.

In the cords from 7 pregnancies complicated by varying degrees of toxaemia, including one premature delivery (36 weeks), the per cent oxygen saturation was shown to present a similar picture to that found in cases of normal pregnancy complicated by cord obstruction:- Vein - 62.3% (31.9 - 79.6); Arteries - 16.1% (10.1 - 24.4). This showed no deficiency in the oxygenation of the blood leaving the placenta and suggested a similar slowing of the umbilical circulation.

Two cases of mild hypertension were included in the study and showed a saturation of 54.5%, 61.2% in

the vein and 13.1%, 42.5% in the arteries.

The delivery anaesthetic in almost every case was a mixture of ½% trilene and air - the remainder having no anaesthetic or a pudendal "block".

Eight cases were delivered by Caesarean section (4 normal, 4 toxaemic pregnancy) and it can be seen that the average oxygen levels were lower than those found at vaginal delivery - particularly those in the toxaemic cases.

The oxygen capacities and therefore the haemoglobin levels were widely scattered throughout but there appears to be a slightly higher level of 17.2 Gms. % in the toxaemic cases as compared with the 15.4 - 16.3 Gms. % found in normal pregnancy.

Walker[33], in addition to his investigation of the effects of pre-eclampsia, has shown variations in per cent oxygen saturation and haemoglobin levels associated with the increasing maturity of a normal pregnancy. Cases were delivered vaginally (easy spontaneous vertex) and by Caesarean section under spinal anaesthesia - no oxygen being administered.

As pregnancy matured there was an increase in the haemoglobin level from 13.9 Gms.% at the 21st week to an average of 15.4 Gms.% at term (Caesarean section deliveries). Thereafter there was shown to be a

further marked increase to an average of 18.3 Gms.% at the 42nd - 43rd week. The average result of a series of haemoglobin estimations at term was 16.5 Gms.%.

The average oxygen saturation in the umbilical vein dropped from 69.4% (66.7 - 74.3) in the 6 premature cases to 56.6% (52.8 - 61.3) at term and then showed a markedly lower level at 42 - 43 weeks of 34.3% (22.6 - 43.8). The oxygen saturation in the umbilical arteries showed a similar trend, the arterio-venous difference remaining at a fairly constant 30 - 35%.

The fall in the oxygen saturation was not entirely due to the increase in haemoglobin level and oxygen capacity. The actual oxygen content in volumes per cent was diminished as pregnancy continued - the average content of the umbilical vein being 13.3 vols.% (premature), 11.7 vols.% (term) and 8.3 vols.% (post-mature).

The results obtained in spontaneous vaginal deliveries at term were somewhat lower - average saturation being:- vein - 53.7%, arteries - 22.0%. These two sets of results are both lower than those of Clemetson but the difference in the oxygen saturation between vein and arteries was similar in all groups.

Of the six cases of pre-eclampsia (34 - 39 weeks) five were delivered by Caesarean section again with spinal anaesthesia. All were classified as severe and the average oxygen saturation was 23.7% (10.5 - 32.5) in the umbilical vein and 7.4% (o.o - 13.2) in the arteries, with a haemoglobin level of 17.8 Gms.% which was much higher than that of normal pregnancies of the same duration. The single case delivered vaginally, without anaesthesia, showed a higher level of oxygen saturation (vein 46.8%; arteries 20.8%) but it was classified as a moderate degree of pre-eclampsia. No indication is given of the duration of the toxaemia.

Haemoglobin levels in the cords from 53 toxaemic pregnancies showed a much higher level than in normal pregnancies particularly if the cases were severe and of long duration.

It was suggested (1) that there was a marked impairment of the oxygenation of the foetal blood, the average arterial level of saturation in the cord being less than one half of that found in normal pregnancy; and (2) that the increase in the haemoglobin level was a response by the foetus to compensate for a degree of anoxia.

RESULTS OF PRESENT STUDY

I. NORMAL PREGNANCY

Spontaneous Vertex Delivery

All the cases (112) in this group had an uneventful pregnancy with a blood pressure recording of less than 130/80, no albuminuria, no more than a slight trace of ankle oedema and no excessive weight gain. All were spontaneous vertex deliveries, chloroform being administered during the birth of the head. All the infants survived. Excluded from this group were all cases in which (1) the cord was round the neck or limb at birth (about one third - one half of all cases attended); (2) the cord was abnormally short and was under tension at delivery; (3) oxygen was administered during labour; (4) heavy sedation was given within two hours of delivery; and (5) there was undue delay or difficulty with the delivery.

The complete tables of results are shown in the appendix to this thesis (Tables A - H) and only the average results, with the number of cases studied, are given in Table III. The cases are classified according to the duration of pregnancy in weeks as calculated to the nearest week from the first day of the last menstrual period. In almost all the cases in this group this was fairly accurately known as the

TABLE III. AVERAGE RESULTS - NORMAL PREGNANCY

Duration of Pregnancy	No. of Cases	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight	Placenta weight	Coefft. of Oxygen Utiln.
		Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb. ozs	lb. ozs	
35	1	17.17	16.2	79.1	9.64	16.4	43.9	32.22	37.66	4 1½	1 1	0.44
36	4	16.82	17.1	73.5	9.06	16.9	40.3	41.06	47.49	6 0	1 5	0.46
37	11	16.61	16.6	74.5	8.82	16.8	39.1	38.82	44.45	6 3¾	1 4¾	0.47
38	10	16.32	17.5	69.7	8.46	17.7	35.8	39.72	45.37	6 13¾	1 3¼	0.50
39	13	15.33	17.7	64.7	7.33	17.7	30.9	40.62	47.07	6 14½	1 5¾	0.52
40	30	14.64	17.2	63.5	7.21	17.3	31.0	41.42	47.23	7 8	1 6½	0.50
41	17	15.57	17.7	65.7	7.50	17.7	31.8	40.43	46.27	7 9½	1 7½	0.52
42	19	12.60	17.7	53.5	4.86	17.7	20.5	41.55	48.10	7 13	1 8	0.62
43	7	13.33	18.2	55.0	4.76	18.3	19.3	42.13	48.09	7 7½	1 5	0.65

majority of the patients were booked primigravidae who had attended the antenatal clinic from the second or third month of pregnancy.

Oxygen content

Studying the oxygen level in volumes per cent in the umbilical vein, Table III shows a gradual fall from 17.17 vols.% in the single case at the 35th week to the average result of 13.33 vols.% in the 7 cases at the 43rd week of gestation, with a marked similarity of the results at 39 weeks (15.33 vols.%), 40 weeks (14.64 vols.%) and 41 weeks (15.57 vols.%). After the 41st week the drop in oxygen content is most marked.

As the results of all investigations tend to show a very similar pattern in the 39, 40, 41 week groups, and for the sake of forming a larger and more comprehensive group for statistical comparison with cases complicated by pre-eclampsia and hypertension, the 60 cases from these three groups have been taken to represent the results to be expected in cases of normal pregnancy at full term.

For the comparison of cases of prematurity and postmaturity with those delivered at term all cases under 39 weeks (26) and over 41 weeks (26) have been grouped respectively as shown in Table IV.

TABLE IV

NORMAL PREGNANCY

Average Results - Premature, Term, Postmature Groups.

Duration of Pregnancy	No. of Cases	Umbilical Vein			Umbilical Arteries			A-V Diffce. %	CO_2 vols.%		Baby weight lb. ozs	Placenta weight lb. ozs	Coefft. of Oxygen Utiln.
		Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%		Umb. Vein	Umb. Arts.			
GROUP A													
35-38	26	16.55 ±1.14	17.0 ±1.1	72.7 ±4.62	8.75 ±0.89	17.2 ±1.2	38.2 ±4.29	34.5	39.30 ±2.67	45.01 ±2.98	6 5¾	1 4	0.48
GROUP B													
39-41	60	15.06 ±1.70	17.4 ±1.4	64.4 ±4.53	7.31 ±1.16	17.5 ±1.4	31.2 ±4.53	33.2	40.96 ±2.93	46.92 ±2.64	7 6½	1 6¾	0.51
GROUP C													
42-43	26	12.80 ±1.60	17.8 ±1.9	53.9 ±5.63	4.84 ±1.51	17.9 ±2.1	20.2 ±6.13	33.7	41.71 ±2.94	48.10 ±2.65	7 11½	1 7¼	0.63

Results

Oxygen content of umbilical vein.

A. Less than 39 weeks (26 cases)

Mean oxygen content = 16.55 vols.% (13.96 - 18.84)

Standard deviation (s) = ± 1.14 vols.%

Standard error of mean (e) = ± 0.224 vols.%

B. 39 - 41 weeks (60 cases)

Mean oxygen content = 15.06 vols.% (10.65 - 18.46)

Standard deviation (s) = ± 1.70 vols.%

Standard error (e) = ± 0.219 vols.%

C. Over 41 weeks (26 cases)

Mean oxygen content = 12.80 vols.% (9.59 - 15.55)

Standard deviation (s) = ± 1.60 vols.%

Standard error (e) = ± 0.314 vols.%

Statistical comparison of groups A and B show that $t = \pm \frac{1.49}{0.313} = \pm 4.76$ which gives a P value of less than 0.01 which is highly significant.

Comparing B and C, $t = \pm \frac{2.26}{0.383} = \pm 5.90$ which again gives a P value of less than 0.01.

It is shown, therefore, that there is a highly significant fall in the average oxygen content of the umbilical vein as pregnancy matures from before the 39th week to full term, with a further marked and

highly significant fall in the oxygen content of the blood reaching the foetus in pregnancies which are prolonged beyond the 41st week.

The results tend on the whole to be higher than those of previous workers.

The average oxygen content of the blood in the umbilical arteries shows a parallel fall as pregnancy continues, from 9.64 vols.% at 35 weeks to 7.33, 7.21, 7.50 vols.% at term with a further drop to 4.76 vols.% at the 43rd week, the arterio-venous difference remaining at a fairly constant 7.50 - 8.50 vols.%.

Considering the three main groups - premature, term and postmature:

A. Less than 39 weeks (26)

Mean oxygen content - 8.75 vols.% (7.27 - 10.68)

Standard deviation (s) - ± 0.89 vols.%

Standard error (e) - ± 0.175 vols.%

B. 39 - 41 weeks (60)

Mean oxygen content - 7.31 vols.% (4.62 - 10.55)

Standard deviation (s) - ± 1.16 vols.%

Standard error (e) - ± 0.150 vols.%

C. Over 41 weeks (26)

Mean oxygen content - 4.84 vols.% (1.84 - 7.89)

Standard deviation (s) - ± 1.51 vols.%

Standard error (e) - ± 0.296 vols.%

Comparing: A & B: $t = \pm \frac{1.44}{0.231} = \pm 6.23$ ∴ $P < 0.01$

B & C: $t = \pm \frac{2.47}{0.332} = \pm 7.44$ ∴ $P < 0.01$

Again a highly significant difference between the groups is shown although the results are, on the whole, higher than those given by previous workers. As can be seen from the tables in the appendix and the above ranges the scatter in all groups was marked with considerable over-lapping from week to week.

Haemoglobin Levels

As stated earlier these were estimated in duplicate and verified in the large majority of cases by oxygen capacity determinations.

Table III and the Appendix show a very marked variation in the haemoglobin levels at all stages of pregnancy. There is no regular pattern of the average results but there appears to be a slight tendency for the average level to rise as pregnancy matures - the average result of the seven cases at 43 weeks being 18.2 Gms.% in the umbilical vein compared with 17.1 Gms.% (36 weeks - 4 cases) and 16.6% (37 weeks - 11 cases).

The statistical comparison of the haemoglobin levels in the umbilical vein of the three main groups: (see Table IV)

A. Under 39 weeks Mean (Gms.%) 17.0 (15.2 - 19.2)
Standard deviation (s) ± 1.1
Standard error (e) ± 0.216

B. 39 - 41 weeks Mean (Gms.%) 17.4 (13.7 - 20.6)
Standard deviation (s) ± 1.4
Standard error (e) ± 0.181

C. Over 41 weeks Mean (Gms.%) 17.8 (13.4 - 22.0)
Standard deviation (s) ± 1.9
Standard error (e) ± 0.373

Comparing A & B: $t = \pm \frac{0.4}{0.282} = \pm 1.42$ ∴ $P = 0.2$

B & C: $t = \pm \frac{0.4}{0.415} = \pm 0.96$ ∴ $P = 0.35$

A & C: $t = \pm \frac{0.8}{0.431} = \pm 1.86$ ∴ $P < 0.1$

No highly significant difference is shown between the haemoglobin levels in the three main groups but the average level in the postmature group may be significantly higher than the average level in the premature group..

The results are similar to those of previous workers quoted earlier (16.5 - 17.9 Gms.%) and show a similar wide range of levels in all groups. The results tend to agree with those of Marks (1955)[53] in showing no significant change with maturity as was

shown by Walker (1953)[33].

Oxygen Saturation

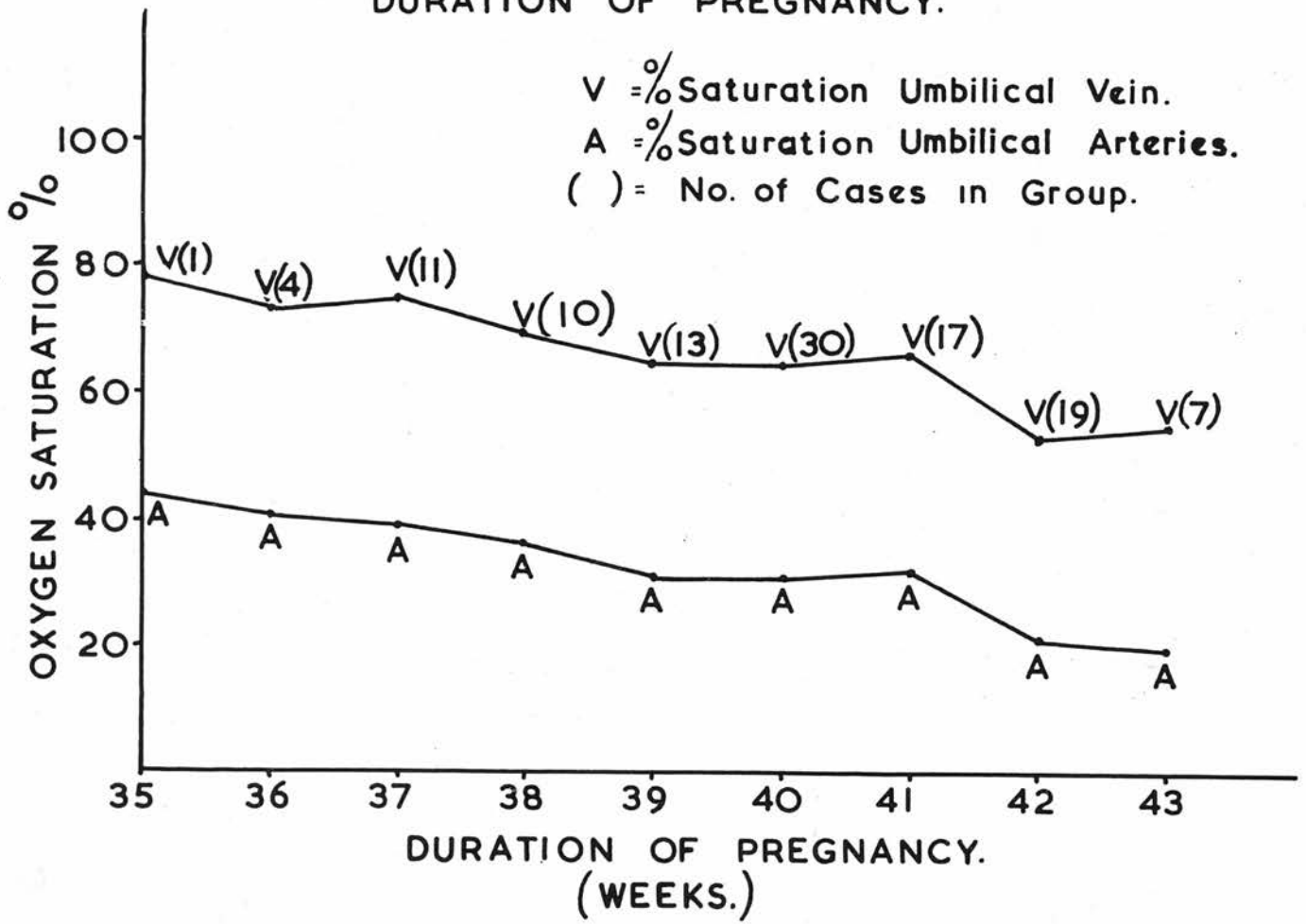
From the average results given in Table III and the graph of them (Fig. I) it will be seen that the per cent oxygen saturation in both the umbilical vein and arteries shows a fairly steady tendency to fall from the earliest case at 35 weeks (V. 79.1%, A. 43.9%) to the average of 10 cases at 38 weeks (V. 69.7%, A. 35.8%). The average results of the cases delivered at 39 (13), 40 (30), and 41 (17) weeks are very similar showing oxygen saturations of 64.7%, 63.5% and 65.7% in the vein and 30.9%, 31.0%, 31.8% in the arteries. Thereafter at 42 (19) and 43 weeks (7) there is a further and more marked drop in the level of saturation in both vessels.

As will be seen from the graph there is a fairly constant difference of 30 - 35% between the saturations in the two vessels. The average differences for the three main groups (Table IV) are A: 34.5%, B: 33.2% and C: 33.7%.

The average results at term were very similar to those of Clemetson (1953)[32] - V. 63.2%, A. 31.5% - and Watts (1951)[31] - V. 66% - but were higher than those of other investigators.

The results showed a wide range with much over-

FIG I. GRAPH SHOWING AVERAGE OXYGEN SATURATION IN THE CORD VESSELS WITH RELATION TO DURATION OF PREGNANCY.



lapping from week to week.

Even as late as the 38th week there is found to be an average oxygen saturation 5% higher than the level at term which is possibly significant and at the 37th week the level is 10% higher. By the 42nd week the average saturation has dropped by 11% - more than twice the standard deviation of the term group of 60 cases (± 4.53%) - see Table IV.

Comparing the three main groups - A, B & C:-

Group A:

Umbilical vein

Mean oxygen saturation %	72.7 (62.4 - 82.2)
Standard deviation (s)	± 4.62
Standard error (e)	± 0.906

Umbilical arteries

Mean oxygen saturation %	38.2 (31.8 - 47.3)
Standard deviation (s)	± 4.29
Standard error (e)	± 0.841

Group B:

Umbilical vein

Mean oxygen saturation % 64.4 (55.7 - 74.9)

(s) = ± 4.53 (e) = ± 0.585

Umbilical arteries

Mean oxygen saturation % 31.2 (20.3 - 45.8)

(s) = ± 4.53 (e) = ± 0.585

Group C:

Umbilical vein

Mean oxygen saturation % 53.9 (37.3 - 64.9)

(s) = ± 5.63 (e) = ± 1.104

Umbilical arteries

Mean oxygen saturation % 20.2 (10.4 - 35.3)

(s) = ± 6.13 (e) = ± 1.202

Umbilical Vein

A & B: $t = \pm \frac{8.3}{1.078} = \pm 7.70$ ∴ $P < 0.01$

B & C: $t = \pm \frac{10.5}{1.249} = \pm 8.41$ ∴ $P < 0.01$

Umbilical Arteries

A & B: $t = \pm \frac{7.0}{1.024} = \pm 6.84$ ∴ $P < 0.01$

B & C: $t = \pm \frac{11.0}{1.337} = \pm 8.23$ ∴ $P < 0.01$

All the differences are highly significant. There is a fall in the level of per cent oxygen saturation in the vein and arteries from the premature group to the term group with a further and more marked drop after the 41st week of pregnancy.

As no significant variation was shown between the average levels of haemoglobin - although a slight trend was apparent - it must follow that the drop in satura-

tion is due very largely to the fall in actual oxygen content of the blood.

Of the 26 cases delivered postmaturely only 4 showed macroscopic infarction of the placenta. The oxygen saturation in the vein was 42 weeks (37.3%, 53.4%), 43 weeks (51.4%, 62.9%). The first result is the lowest recorded. Labour was induced in only 2 cases - by pitocin drip - and the saturation was 51% and 55% in the umbilical vein. These levels are well within the range of results obtained in cases in which labour was not induced.

Carbon Dioxide Content

The carbon dioxide content of the umbilical vessels at all stages of pregnancy showed a fairly wide scatter of results with a reasonably constant difference of about 5 - 6 vols.% between the levels of the vein and arteries. On inspection of Tables III and IV there appears to be a slight tendency for both levels to rise as pregnancy matures.

Comparative results:

Group A:

Vein Mean CO_2 content (vols.%) - $\frac{39.30}{(33.22 - 43.35)}$

(s) = ± 2.67 (e) = ± 0.523

Arteries Mean CO_2 content (vols.%) - $\frac{45.01}{(37.66 - 50.38)}$

(s) = ± 2.98 (e) = ± 0.584

Group B:

Vein Mean CO_2 content (vols.%) - $\frac{40.96}{(36.21 - 47.85)}$

(s) = ± 2.93 (e) = ± 0.378

Arteries Mean CO_2 content (vols.%) - $\frac{46.92}{(40.67 - 52.71)}$

(s) = ± 2.64 (e) = ± 0.341

Group C:

Vein Mean CO_2 content (vols.%) - $\frac{41.71}{(34.89 - 47.37)}$

(s) = ± 2.94 (e) = ± 0.577

Arteries Mean CO_2 content (vols.%) - $\frac{48.10}{(44.18 - 52.82)}$

(s) = ± 2.65 (e) = ± 0.520

Umbilical Vein

A & B: $t = \pm \frac{1.66}{.645} = \pm 2.57$ ∴ $P < 0.02$

B & C: $t = \pm \frac{0.75}{.690} = \pm 1.09$ ∴ $P = 0.3$

Umbilical Arteries

A & B: $t = \pm \frac{1.91}{.676} = \pm 2.83$ ∴ $P < 0.01$

B & C: $t = \pm \frac{1.18}{.622} = \pm 1.90$ ∴ $P > 0.05 < 0.1$

It is shown that the average carbon dioxide content of the cases delivered at term is significantly higher than that of the premature group but the difference post term is probably not significant.

These results and the fairly constant difference of 5 - 6 vols.% between the vein and arteries are similar to those found by Eastman (1932)[24] and Clemetson (1953)[32].

Other Records

The average weights of infants and placentae show the expected increases on the whole although there is wide variation in each group. There is also a slight drop shown in the ratio of placental weight to baby weight from 0.22 at 36 weeks to 0.17 at 43 weeks as would be expected from the growth of the foetus.

The values for the coefficient of utilisation, which give an indication of how much available oxygen is being used by the foetus, show a definite rise. This is expected as it has been shown that increasingly less oxygen becomes available while the A-V difference remains constant.

II. NORMAL PREGNANCY

Spontaneous Vertex Delivery

Cord Round Neck (Appendix-Table I)

Seven cases from normal pregnancy (chloroform anaesthesia), where the cord was found at delivery to be round the neck of the foetus at least once, were investigated. Six cases were at term (39 - 41 weeks) and the other at 36 weeks. Six of the seven infants survived but three were very shocked and required resuscitation; in one of these cases there were signs of foetal distress in utero - staining of the liquor with meconium and a slowing of the foetal pulse rate. This was the premature infant which later died; post mortem showing intra-ventricular haemorrhage and kernicterus. The oxygen saturation of the blood in the umbilical vein was 46.1%, and in the arteries 15.0% - both far below normally expected levels in premature pregnancy (72.7%, 38.2%).

The average results in the remaining six cases compared with the average results of the normal term group without cord obstruction were:

Umbilical Vein	AVERAGE	RANGE	NORMAL, NO OBSTRUCTION
Oxygen content (vols.%)	13.33	(4.11 - 20.35)	15.06
Haemoglobin (Gms.%)	17.3	(14.9 - 20.3)	17.4
Oxygen saturation (%)	56.1	(20.6 - 74.8)	64.4

Umbilical Vein Contd.	AVERAGE	RANGE	NORMAL, NO OBSTRUCTION
Carbon dioxide cont. (vols.%)	42.86	(37.67 - 46.31)	40.96
Umbilical Arteries			
Oxygen content (vols.%)	2.68	(2.13 - 4.09)	7.31
Haemoglobin (Gms.%)	17.0	(14.3 - 18.8)	17.5
Oxygen saturation (%)	11.7	(8.5 - 16.7)	31.2
CO_2 content (vols.%)	51.15	(47.24 - 54.57)	46.92

The oxygen saturation in the vein shows a very wide range but the average result is 8% lower than that found in non-obstructed cases - partly due to the one very low saturation of 20.6% from a case where the baby was very shocked at birth, the cord having been twice tightly round the neck.

The oxygen saturation is very much lower than normal in the arteries, the average result being 20% lower. This makes a greater difference (45%) between the levels in the two vessels.

The carbon dioxide levels again showed a wide range but were, on the whole, higher than normal with a difference of 8 vols.% between the vessels as

compared with the normal 6 vols.%.

These results are similar to those of Clemetson[32] showing the increased difference in oxygen and carbon dioxide levels between the vein and arteries.

The group is much too small for statistical comparison and has been included to show (1) that if the cord is obstructed in any way the oxygen reserve is diminished as the circulation rate is slowed and (2) why all cases of potential cord obstruction were not included in the normal comparative study.

Delayed Delivery

Three cases of undue delay during the second stage are shown in the same table (I), again to show the lower levels of oxygen saturation. In one case there was an interval of about 30 minutes while the head was on the perineum and in the other two considerable difficulty in delivering the shoulders was encountered. The average oxygen saturations were:- vein 48.5%, arteries 14.8%. Two of the infants were in poor condition at birth and required resuscitation.

III. TWIN PREGNANCY

Vertex and Breech Delivery

The cord bloods from four twin pregnancies were studied, blood samples being obtained from the cords of seven infants. All the pregnancies were normal, showing no signs or symptoms of pre-eclampsia apart from slight ankle oedema. All were premature (33 - 38 weeks) deliveries - 4 vertex, 3 assisted breech. Chloroform anaesthesia was used in all cases.

Complete results are given in the Appendix (Table J). Average results were:-

	(4) VERTEX	(3) BREECH	(7) TOTAL	
Umbilical Vein				
Oxygen content (vols.%)	9.22	8.94	9.10	(6.31 - 12.89)
Haemoglobin (Gms.%)	16.8	17.6	17.1	(14.0 - 20.0)
Oxygen saturation (%)	41.0	37.9	39.7	(25.7 - 54.4)
CO_2 content (vols.%)	41.01	43.57	42.11	(34.27 - 45.30)
Umbilical Arteries				
Oxygen content (vols.%)	4.86	4.38	4.65	(2.20 - 8.20)
Haemoglobin (Gms.%)	16.8	18.1	17.4	(14.6 - 19.8)
Oxygen saturation (%)	21.7	18.1	20.1	(9.1 - 35.6)
CO_2 content (vols.%)	45.65	47.85	46.59	(38.25 - 49.52)

The results of oxygen content and per cent saturation are much lower than in cases of single pregnancy, particularly premature normal pregnancy where the average oxygen saturation was shown to be 72.7% in the vein and 38.2% in the arteries. Both of these figures are outside the ranges found in the twin pregnancies.

The levels in the cases of breech delivery are lower on the whole than those of the vertex deliveries.

The difference in oxygen saturation between the blood reaching the foetus and that leaving it is 20% (compared with the normal 30-35%) probably due to the fact that foetal requirements were less - average weight being about 4¼ lbs.

The haemoglobin range is wide but similar to that found in single pregnancies. The level tends to be slightly higher in the cases of breech delivery.

The above results are probably significantly different from those found in normal single pregnancy of the same duration but there are too few cases for statistical analysis.

It is an opportune time to mention a few breech deliveries from normal pregnancy. Owing to the variable and usually prolonged anaesthesia, the different methods of delivery and the high incidence

of potential, if not true, cord obstruction only five cases at term were studied and the average oxygen saturation was found to be vein - 54.5%, arteries - 18.7%, both levels being much lower than those found at spontaneous vertex deliveries.

IV. PREGNANCY COMPLICATED BY PRE-ECLAMPSIA

Spontaneous Vertex Delivery

In this group of 38 cases - all spontaneous vertex deliveries - chloroform was administered with the birth of the head in all except three of the very premature cases. All had a blood pressure reading of 134/82 or more and showed some degree of oedema, with or without albuminuria. All the infants survived.

Again all cases of cord obstruction were excluded and no patients received oxygen during labour but four of the premature severe cases had fairly heavy sedation during labour - luminal, pethidine, heroin, etc.

As can be seen from Tables K and L in the Appendix, which give full details, several deliveries were induced surgically and by pitocin drip.

Not all were booked cases - about 10 were sent into hospital because of toxaemia - so the duration of the pregnancy and toxaemia was not known for certain in all cases.

All the placentae were examined for macroscopic infarction.

The cases have been divided, as were the normal pregnancies, into three main groups: A - less than 39 weeks; B - 39 - 41 weeks; and C - over 41 weeks.

Group B. 39 - 41 weeks (23 cases) Appendix-Table K.

This main group of 23 cases has been subdivided into 4 main groups:

(1) 4 cases showing generalised oedema and excessive weight gain, with no albuminuria (except in one case in labour) and a blood pressure reading not exceeding 140/90.

(2) 5 cases of mild pre-eclampsia with minimal albuminuria, a blood pressure of up to 150/100 and some oedema.

(3) 10 cases of moderate pre-eclampsia all showing an elevation of blood pressure, albuminuria and oedema.

(4) 4 cases of severe pre-eclampsia, one of whom had a fit 11 hours postpartum.

Induction:- Mild cases: one induced by pitocin drip. Moderate cases: one medical induction with pituitrin and three surgical inductions - rupture of membranes. Severe cases: one admitted in labour, one pitocin drip, one surgical induction and one pitocin drip and surgical induction.

Table V shows the average results of the four groups and it will be seen that there is a progressive fall in the oxygen content and saturation of the blood in the umbilical vein and, to a less uniform extent, in the arteries, as the severity of the toxaemia

TABLE V. AVERAGE RESULTS - TOXAEMIA 39 - 41 WEEKS

No. of cases	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight	Placenta weight	Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb. ozs	lb. ozs	
GROUP 1.	GENERALISED OEDEMA - EXCESSIVE WEIGHT GAIN										
4	12.34	17.1	54.3	4.75	16.9	21.0	42.35	48.57	7 8	1 8½	0.62
GROUP 2.	MILD PRE-ECLAMPSIA										
5	12.57	17.7	53.0	3.92	17.9	16.4	41.80	47.07	8 0	1 6¾	0.69
GROUP 3.	MODERATE PRE-ECLAMPSIA										
10	12.00	18.1	51.0	4.67	18.1	19.5	43.74	50.18	7 9¼	1 8¾	0.61
GROUP 4.	SEVERE PRE-ECLAMPSIA										
4	10.47	18.4	42.8	4.14	18.3	17.0	40.23	46.02	6 7	1 4	0.62
	ALL CASES										
23	11.92	17.9	50.6	4.43	17.9	18.7	42.47	48.50	7 7½	1 7½	0.63
ST. DEV	±2.21	±1.35	±8.89			±5.79	±3.85				

increases.

There is a corresponding rise in the average haemoglobin levels from 17.1 Gms.% in the Group (1) to 18.4 Gms.% in the severe cases.

Most of the individual groups are too small for statistical analysis but even in Group (1) the highest oxygen saturation recorded was 59.2% which is 5% lower than the average of the normal group at term (64.4%).

The difference between the oxygen saturation in the vein and arteries is (1) 33.3%; (2) 36.6%; (3) 31.5% and (4) 25.8% - showing a slight tendency to diminish with the increasing severity.

The average carbon dioxide content of the vein and arteries shows the usual wide range of results with no obvious change with increasing severity except in the moderately severe group where it is slightly higher than the normal average (40.96 vols.%). There is a difference of about 6 vols.% between the two levels in all the groups.

The average baby weight is lower in the severe cases. This may account for the lower arterio-venous difference.

The coefficient of oxygen utilisation is variable but all the average results are higher than in the

normal cases (0.51), less oxygen being available for the foetus, with a diminished oxygen reserve.

Gross infarction of the placenta occurred in only 6 cases: (1) 1; (2) 1; (3) 2; (4) 2. In the cases showing infarction the oxygen saturations in the umbilical vein were: (1) 56.9%; (2) 48.5%; (3) 47.5%, 29.5%; (4) 26.8%, 33.6%. Apart from the first result this accounted for the lowest levels in each group.

With regard to the method of induction of labour the following oxygen saturations in the vein were found:-

(a) Surgical induction

Group (3) 54.8%, 52.4%, 59.2%

Group (4) 59.6%.

All high levels of saturation.

(b) Pitocin drip induction

Group (2) 48.5%.

" (4) 33.6%.

(c) Both methods

Group (4) 26.8%.

Oxygen saturation was much lower in the cases in which a pitocin drip was given but two of these cases were in the severe group (4).

For statistical comparison with normal cases, the

entire group of 23 cases was used, the average results being:-

Umbilical Vein	AVERAGE	(s)	RANGE	NORMAL TERM AVERAGE
Oxygen content (vols.%)	11.92	±2.21	(6.54 - 15.38)	15.06
Haemoglobin (Gms.%)	17.9	±1.35	(14.6 - 19.9)	17.4
Oxygen saturation (%)	50.6	±8.89	(26.8 - 59.6)	64.4
CO_2 content (vols.%)	42.47	±3.85	(34.64 - 47.62)	40.96
Umbilical Arteries				
Oxygen saturation %	18.7	±5.79	(6.8 - 26.0)	31.2

Statistical Comparison

Umbilical Vein

Oxygen content: $t = \pm \frac{3.14}{.510} = \pm 6.16$ ∴ $P < 0.01$

Haemoglobin: $t = \pm \frac{0.5}{.334} = \pm 1.50$ ∴ $P = 0.1$

Oxygen saturation: $t = \pm \frac{13.8}{1.944} = \pm 7.10$ ∴ $P < 0.01$

CO_2 content: $t = \pm \frac{1.51}{.888} = \pm 1.70$ ∴ $P = 0.1$

Umbilical Arteries

Oxygen saturation: $t = \pm \frac{12.5}{1.342} = \pm 9.31$ ∴ $P < 0.01$

There is therefore a highly significant difference in the oxygen content and per cent saturation in the

cord bloods of the two groups, those in the toxaemic cases being significantly lower than in the normal term group.

There is possibly a significant change in the carbon dioxide contents and haemoglobin levels, both being higher in the toxaemic cases. When only the 14 cases of moderate and severe toxaemia are included, the average haemoglobin is 18.2 ± 1.2 Gms.% which is significantly higher than the normal group ($t = \pm \frac{0.8}{.369} = \pm 2.17$, $P < 0.02$)

Taking into consideration the duration of the toxaemia and ignoring the severity of the signs and symptoms, we have 6 cases of a duration of less than three weeks - all booked cases attending the antenatal clinic. Three were mild cases. The individual results, with the grouping as to severity, of the oxygen saturation in the umbilical vein were:-

Group (1) - No. 167 - 59.2%.

Group (2) - No. 136 - 55.3%; No. 152 - 53.1%.

Group (3) - No. 176 - 52.5%; No. 121 - 59.2%.

Group (4) - No. 130 - 59.6%.

All are high levels in each group. The last (No. 130) was the case admitted with severe toxaemia and induced immediately - she had attended the clinic three weeks before admission and gave a history of symptoms of

1-2 weeks duration. None of the placentae showed gross infarction.

The average per cent saturation in these cases was 56.5%, compared with an average of 48.5% in the remaining 17 cases which were of a duration of 3 weeks or more. Arteries:- 20.6% (under 3 weeks), 18.0% (3 weeks or more).

Although the former group is very small there appears to be a significant difference between the two levels of oxygen saturation. ($t = \pm \frac{8.0}{2.692} = \pm 2.97$ $\therefore P = 0.02$). Both groups show a significantly lower oxygen saturation than normal.

Average haemoglobin levels were 17.5 Gms.% in the short duration (6), 18.0 Gms.% in those of longer duration (17).

There is a significantly lower average level of oxygen saturation and content in the umbilical vein and arteries in cases of pre-eclampsia which appears to be dependent not only on the severity of the toxaemia but also on its duration.

There is a significant rise in haemoglobin level in all but mild cases of pre-eclampsia with a probably significant increase in the carbon dioxide contents of both vessels (difference still about 6 vols.%).

Group A: Less than 39 weeks (9 cases) Appendix-Table L.

In this group are included, as might be expected, the severe cases of pre-eclampsia which were terminated at an early date. This accounted for 4 cases (33 - 36 weeks) in which labour was induced surgically, and all of which showed macroscopic placental infarction.

The remaining 5 cases (38 weeks) were of moderate or severe pre-eclampsia apart from one case of mild toxaemia further complicated by being Rhesus negative. In the last, labour was induced surgically because of the Rhesus complication (No. 53).

The average results of the four severe cases were:-

Umbilical Vein

Oxygen content (vols.%)	11.03	(8.53 - 13.42)
Haemoglobin (Gms.%)	18.2	(16.4 - 19.4)
Oxygen saturation (%)	45.1	(36.6 - 52.7)
CO_2 content (vols.%)	42.93	(36.98 - 47.98)

Umbilical Arteries

Oxygen saturation (%)	18.3	(15.0 - 23.8)

Although this group is very small it is shown that there is a markedly lower level of oxygen content and saturation in the umbilical vein and arteries with a difference in saturation of 27%. The average haemoglobin level is much higher than that found in

normal premature cases (17.0 Gms.%).

All were of a duration of three weeks or more.

One case had a pitocin drip as well as a surgical induction - the oxygen saturation being:- vein - 38.8%, arteries - 16.9%.

Total group (9 cases) compared with the average results of normal premature pregnancy are as follows:-

Umbilical Vein	AVERAGE	RANGE	NORMAL PREMATURE
Oxygen content (vols.%)	11.95	(8.53 - 13.42)	16.55
Haemoglobin (Gms.%)	17.6	(12.8 - 19.9)	17.0
Oxygen saturation (%)	51.2	(36.6 - 69.6)	72.7
CO_2 content (vols.%)	42.06	(36.98 - 47.98)	39.30
Umbilical Arteries			
Oxygen saturation (%)	21.2	(15.0 - 28.5)	38.2

There is a highly significant difference in the average oxygen content and saturation in the umbilical vein compared with normal premature pregnancy.

Oxygen content: $t = \pm \frac{4.60}{.622} = \pm 7.40$ $\therefore P < 0.01$

Oxygen saturation: $t = \pm \frac{21.5}{3.355} = \pm 6.41$ $\therefore P < 0.01$

The difference in saturation between the vein and

arteries remained at a fairly constant 30% except for the severe cases where it was a little lower (smaller infants).

Excluding case 53 (Rhesus negative) where the haemoglobin level was 12.8 Gms.%, the average haemoglobin was 18.2 Gms.% which is significantly higher than that in the normal premature group ($t = \pm \frac{1.2}{.476} = \pm 2.52$: $P = 0.05$).

The rise in carbon dioxide content is probably also significant ($t = \pm 2.13$: $P < 0.1$).

All cases were of a duration of three weeks or more apart from No. 53 which was not really comparable but showed the one high level of oxygen saturation in the vein (69.6%) - due largely to the very low haemoglobin level associated with the additional complication.

On the whole the average results are similar to those found in the "term" group of toxaemic cases.

Appendix Table L also shows one case of pre-eclampsia superimposed on essential hypertension (B.P. $\frac{170}{100}$ at 11th week) - delivered at 33 weeks - cord round neck at delivery. Oxygen saturation:- Vein 61.0%; Arteries 19.1%. In this case labour was induced by artificial rupture of the membranes and pitocin drip.

Group C: Over 41 weeks (6 cases) Appendix-Table L

The cases in this group were all fairly mild

cases of pre-eclampsia apart from one unbooked case (155) in which labour was induced surgically on the day following admission. 5 of the placentae showed macroscopic infarction. All babies survived.

The average results compared with normal pregnancy over 41 weeks were:-

Umbilical Vein	AVERAGE	RANGE	NORMAL POSTMATURE
Oxygen content (vols.%)	11.05	(8.09 - 13.74)	12.80
Haemoglobin (Gms.%)	18.4	(15.7 - 21.0)	17.8
Oxygen saturation (%)	45.0	(35.1 - 56.3)	53.9
CO_2 content (vols.%)	41.11	(39.78 - 44.29)	41.71
Umbilical Arteries			
Oxygen saturation (%)	14.6	(6.2 - 21.5)	20.2

Even in the mild cases in which pregnancy was prolonged over 41 weeks there is a further fall in oxygen content and saturation of the umbilical vein - the average results being lower than in any group previously recorded apart from the four cases of severe toxaemia at term (10.47 vols.%: 42.8% saturation).

There is a high average haemoglobin and a slightly lower level of carbon dioxide in the vein than was found in the premature and term cases of toxaemia.

As will be seen above, the oxygen levels appear to be even lower than the corresponding levels in normal postmature pregnancy - while the haemoglobin is slightly higher. The arterio-venous difference is again about 30%.

The group is too small for statistical analysis.

The case in which labour was induced surgically showed an oxygen saturation of 54.4% in the vein.

V. PREGNANCY COMPLICATED BY HYPERTENSION

In this small group (8 cases) all were delivered at 39 - 41 weeks (Appendix-Table M) - spontaneous vertex deliveries, chloroform anaesthesia and no potential cord obstruction. The earliest recorded blood pressure was at least $\frac{130}{80}$, rising to $\frac{145}{95}$ - $\frac{180}{100}$ and there was no albuminuria or oedema. All the infants survived. Two of the placentae showed macroscopic infarction. The majority of cases showed a very mild degree of hypertension.

Comparing the average results with those of normal pregnancy (39 - 41 weeks):-

	AVERAGE	RANGE	AVERAGE NORMAL
Umbilical Vein			
Oxygen content (vols.%)	17.11	(15.30 - 18.92)	15.06
Haemoglobin (Gms.%)	18.0	(15.8 - 19.6)	17.4
Oxygen Saturation (%)	71.1	(67.3 - 76.7)	64.4
CO_2 content (vols.%)	41.68	(37.10 - 44.69)	40.69
Umbilical Arteries			
Oxygen saturation (%)	34.8	(28.8 - 39.1)	31.2

There is a higher average oxygen content and saturation in the vein and arteries than that found in normal pregnancy - in the case of the vein all the

individual results are higher than the average normal.

The number is very small for statistical analysis, but by applying the usual test the increase in oxygen level appears to be significant. (Oxygen content: vein $t = \pm 4.05$ $\therefore P < 0.01$. Oxygen saturation: vein $t = \pm 5.0$ $\therefore P < 0.01$; arteries $t = \pm 2.39$ $\therefore P = 0.05$).

The difference in saturation between the vein and arteries is 36% - slightly higher than that in normal cases.

The change in Haemoglobin levels ($t = \pm 1.40$) and carbon dioxide contents ($t = \pm 0.77$) is probably not significant.

The 2 cases showing placental infarction had an oxygen saturation in the vein of 74.7%, 76.7%.

In 1 case labour was induced surgically - saturation in the vein being 68.7% - well within the range of results obtained.

Table M also gives the results of one case delivered at 37 weeks. Here the oxygen saturation was even higher - vein 80.8%, arteries 45.9%.

The group is exceedingly small and it would be of value to study such cases more fully before drawing any definite conclusions.

VI. FORCEPS DELIVERIES

The cord bloods from fifteen forceps deliveries were investigated. In view of the variations in (a) the indication for the instrumental delivery, (b) the type and degree of anaesthesia used, (c) the anaesthetist, (d) the induction of anaesthesia - delivery interval and (e) difficulty encountered during delivery - no attempt is made at statistical analysis or comparison. The results are included merely to show some of the oxygen levels of the blood reaching the foetus immediately after an instrumental delivery. Table N in the Appendix gives more complete results.

A. Normal Pregnancy

5 cases had a normal pregnancy - apart from one Cardiac (Grade IIA). All were delivered under general anaesthesia at 40 - 41 weeks. Case No. 27 - (Cardiac) had oxygen administered during labour. All had Flaxedil, cyclopropane and oxygen with the addition of nitrous oxide in case No. 21.

		OXYGEN SATURATION %	
INDICATION	CASE NO.	Vein	Arteries
Foetal distress	21	31.3	9.0
"	27	47.8	23.4
"	35	75.1	11.6
Uterine Inertia (cord round neck)	33	81.8	-
Deep transverse arrest	43	43.1	19.2
Average:-		55.8	15.8

The results are very variable but on the whole the oxygen saturation tends to be lower than that found at spontaneous vertex deliveries. The very low reading (31.3%) was associated with foetal distress and the administration of nitrous oxide which was shown by Eastman[25](1936), Smith[29](1939) and Taylor [55](1951) to produce lower than normal oxygen levels if given in quantity. The high reading of 81.8% in the vein was associated with cord obstruction - cord tightly round the neck - no sample obtained from arteries.

In case No. 27, although oxygen had been administered during labour, the saturation level was much lower than normal. This infant showed signs of distress "in utero" and was very limp at delivery - saturation 47.8% vein.

B. Toxaemic Pregnancy

9 cases of varying degrees of toxaemia (37 - 42 weeks) were delivered under various types of general anaesthesia for various reasons:-

INDICATION	DEGREE OF TOXAEMIA	CASE NO.	OXYGEN SATURATION %	
			Vein	Arteries
Foetal distress	moderate	10	41.0	10.8
Second Stage delay	"	16	12.7	8.5
"	"	15	48.0	16.8
Persistent occipito-posterior	mild	48	61.4	-

INDICATION	DEGREE OF TOXAEMIA	CASE NO.	OXYGEN SATURATION % Vein	Arteries
Deep transverse arrest	moderate	113	42.0	13.7
Inertia	mild	58	37.9	8.5
Maternal distress	moderate	37	31.5	14.6
"	mild	64	70.5	31.9
Fulminating intra-partum toxaemia	severe	25	50.5	20.8
AVERAGE (9)			43.9	15.7
AVERAGE NORMAL S/D (39 - 41 weeks)			64.4	31.2
AVERAGE TOXAEMIA S/D (39 - 41 weeks)			50.6	18.7

These results again show a very wide variation but are lower than those found at spontaneous delivery - average saturation being 43.9% which is almost as low as that found in the group of severe cases of toxaemia delivered spontaneously (42.8%) although only one case was placed in that category.

There are too few cases of assorted types to draw any accurate or comparable picture but even these few results do tend to show that at birth much less oxygen is available to the foetus in most instances and consequently, as the arterio-venous difference in saturation remains at about 30%, there is a diminished oxygen reserve. Whether the low levels are due to

the method of delivery or some factor in the anaesthesia or both - there is much more risk of foetal anoxia should any additional complication such as cord obstruction or undue delay occur.

VII. CAESAREAN SECTION DELIVERIES

This group consisted of 38 cases (13 normal pregnancies, 10 complicated by pre-eclampsia, 1 by hypertension, 1 by chronic nephritis and 13 by diabetes mellitus.

All the results are given in full in the Appendix-Tables O, P, Q, and in the following paragraphs some will be summarised. Again a considerable number of variable factors (indication, anaesthetic, type of section etc.) complicate the picture and prevent true comparisons.

(a) Normal Pregnancy: 13 cases. Appendix-Table O.

(1) Elective (10 cases)

7 cases (39 - 41 weeks pregnancy) - all lower segment operations - were delivered under general anaesthesia (Flaxedil, cyclopropane and oxygen in all, with the addition of Pentothal and nitrous oxide in 3 cases).

Average results and ranges of this group:-

Umbilical Vein

Oxygen content (vols.%)	7.90	(6.07 - 15.62)
Haemoglobin (Gms.%)	16.4	(14.0 - 18.2)
Oxygen saturation (%)	35.6	(21.2 - 64.1)
CO_2 content (vols.%)	51.01	(42.64 - 56.40)

Umbilical Arteries

Oxygen saturation (%) 13.8 (8.3 - 31.7)

With the exception of one case (No. 175 - previous C/S and placenta praevia III) - showing per cent oxygen saturation of 64.1% (vein) and 31.7% (arteries) all the results showed an oxygen saturation of less than 41% in the vein. In the cases in which the oxygen saturation was less than 30% in the vein the infants were found to be very shocked at birth. This might be expected as it has been shown that the normal difference in saturation between the vein and arteries is in the order of this figure.

The haemoglobin levels are in the normal range.

The average carbon dioxide content is much higher than at vaginal delivery in both vessels but shows a wide range of results.

Two cases - also lower segment - were postmature. No. 237 (general anaesthesia) showed an oxygen saturation of 33.7% (vein) 12.2% (arteries). Case No. 189 - a known postmature case of 44 weeks in which two pitocin drips had failed to induce labour - was delivered under local anaesthesia. Little liquor was found and this was deeply coloured yellow. Baby weight 9 lb. 9¾ ozs. The oxygen saturation was 37.4%

(vein), 8.6% (arteries), but the haemoglobin level, 16.9 Gms.% was well within the normal range. The low saturation was due to a low oxygen content in the blood and not to an increased oxygen capacity.

One case at term was delivered by classical section under general anaesthesia and the oxygen saturation was found to be similar to that found at spontaneous delivery:- 57.8% (vein), 31.9% (arteries).

(2) In Labour (3 cases)

3 cases of inertia, cervical dystocia and trial of labour were delivered by lower segment section (2 general anaesthesia, 1 spinal and oxygen). The oxygen saturations (V - vein; A - arteries) were:-

No. 30	38 weeks	G.A.	V 68.3%	A 49.5%
No. 88	40 weeks	Spinal	V 73.7%	A 41.5%
No.163	41 weeks	G.A.	V 35.3%	A 7.4%

In the last case (163) the baby was very shocked at birth.

The average saturation in all 13 cases was 42.7% (V); 19.1% (A), which is well below the average result found at spontaneous vaginal delivery but the results were variable and three of the cases were found to have normal levels.

The type and degree of anaesthesia may be the deciding factor, as high and low results were found in elective and non-elective cases but there is a distinct possibility that the variations in oxygen level are largely dependent also on the state of uterine tone at the precise moment of delivery - not always a reasonably constant factor at Caesarean section.

(b) Toxaemic Pregnancy: 10 cases. Appendix-Table P.

(1) One case of fulminating toxaemia (35 weeks) was delivered by classical section under general anaesthesia. The oxygen saturation was 50.7% (V); 15.4% (A) - lower than normal premature pregnancy vaginal delivery. Haemoglobin (18.9 Gms.%) was at a high level. The infant (3 lbs. 8½ ozs.) later died - necropsy showing intrapulmonary haemorrhage.

(2) The nine remaining cases were delivered by lower segment section.

A:- 4 cases were not in labour. Of these, two were delivered because of severe toxaemia at 32 weeks under spinal anaesthesia. The oxygen saturations found were (V) 37.7%; 23.5%: (A) 14.1%; 6.7% - all very low levels with very little oxygen returning from the foetus in either case - one infant died - necropsy

showing hyaline membrane. The haemoglobin levels in both cases were high, 17.9 Gms.%, 19.9 Gms.%. The other two cases had mild pre-eclampsia and the sections were performed for (1) previous section (2) placenta praevia (cardiac) - one spinal and one general anaesthesia. The oxygen saturation was higher in both cases (V) 50.5%; 46.9%: (A) 24.5%; 14.3%.

B:- 5 cases were in labour - 4 mild toxaemia and 1 severe.

In the case of severe toxaemia (40 weeks) the membranes had been ruptured artificially and during labour there were signs of foetal distress and so section was carried out, under general anaesthesia. The oxygen saturations were very low (V - 18.8%; A - 6.4%) and the baby was very limp at birth.

The 4 cases of mild toxaemia (38 - 42 weeks) had all been in labour over 30 hours - all were delivered under general anaesthesia. The oxygen saturations were:-

Vein	33.3%;	58.6%;	13.9%;	26.1%.
Arteries	4.9%;	25.4%;	4.3%	9.1%.
Weeks	38	41	42	42

all very low - particularly the two postmature cases. There were signs of foetal distress only in the case

delivered at 38 weeks (33.3% V) but the two infants from the postmature cases were very limp at birth.

C:- One case of mild hypertension and previous section had an elective section at 39 weeks. The cord was found to be round the baby at birth. Oxygen saturation V 22.1%; A 12.0%.

D:- One case of chronic nephritis (2 previous sections) was also delivered at 39 weeks under general anaesthesia. Oxygen saturation was again found to be very low - V 23.6%; A 10.1%.

As was pointed out earlier there are too many variables to form any definite conclusions but in all the severe cases of pre-eclampsia the levels of saturation were very low indeed - even lower than at vaginal delivery where the lowest saturation found was V 26.8% in one severe case at term. Perhaps therefore the operation or some feature of it is exposing the foetus to an even greater danger of anoxia. Although it is not suggested that the levels found are indicative of the oxygenation of the foetal circulation "in utero" it is shown that the infants at birth are frequently being exposed, however momentarily, to a very low supply of oxygen which may endanger life if prolonged.

(c) Pregnancy complicated by Diabetes Mellitus
13 cases Appendix-Table Q.

The cords from 13 cases complicated by diabetes mellitus were studied. All were delivered prematurely (35 - 38 weeks) but variables were introduced with the type of section and anaesthesia and 3 cases were further complicated by having some degree of pre-eclampsia.

(1) Lower Segment Section (10 cases)

7 cases with no signs or symptoms of toxaemia were delivered (35 - 38 weeks) under spinal anaesthesia with the addition of oxygen in two cases. The results showed a very wide range of both oxygen and carbon dioxide levels - the average results and ranges were:-

Umbilical Vein

Oxygen content (vols.%)	7.36	(2.46 - 13.06)
Haemoglobin (Gms.%)	16.7	(15.2 - 18.4)
Oxygen saturation (%)	33.0	(11.0 - 60.2)
CO_2 content (vols.%)	48.70	(44.07 - 54.48)

Umbilical Arteries

Oxygen saturation (%)	12.2	(2.8 - 24.5)

The haemoglobin levels were all within the normal range and the variation in oxygen saturation was due to the oxygen content alone.

The two cases in which oxygen was administered showed an oxygen saturation of 60.2% and 11.0% in the vein but in the latter case there was some delay in delivery - the operator waiting for a uterine contraction to cease before removing the child.

One case with the cord twice round the neck accounted for another low saturation V 13.6%.

Only three of the cases showed a saturation of over 30% in the vein at delivery.

One infant (35 weeks) 9 lb. 15 ozs. - from a case showing marked hydramnios - died of renal thrombosis.

3 cases showed in addition a mild degree of pre-eclampsia. All were at 36 weeks and delivered under spinal anaesthesia. All the oxygen levels in this group were very low:-

Vein	11.0%;	31.0%;	44.6%.
Arteries	3.5%;	2.5%;	6.3%.

(2) Classical Section (3 cases)

Three cases (35 - 37 weeks) were delivered by classical Caesarean section. One case had spinal anaesthesia and showed an oxygen saturation of V 59.8%; A 18.6%.

The two remaining cases had general anaesthesia (Flaxedil, Pentothal, cyclopropane, nitrous oxide and

oxygen).

The results (No. 63 - V 71.6%; A 45.8%)
(No.174 - V 34.2%; A 3.9%)

show markedly different levels - the only similarity being the arterio-venous difference.

All three results were above the 30% saturation level required by the foetus.

Again it is impossible to draw any definite conclusions but it is possible to show that almost half of the infants were receiving blood which was less than 30% saturated with oxygen at the moment of birth and so might be in great danger of anoxia.

From the few cases studied, the results obtained at classical Caesarean section appear to be higher on the whole than those of lower segment deliveries - as was also found in normal and toxaemic groups (normal (1 case) V 50.7%, toxaemic (1 case) V 57.8%, diabetic (3 cases) V 55.2%).

SUMMARY OF RESULTS

As the main difference shown throughout has been in oxygen levels of the cord and to some extent in haemoglobin levels, the average figures for each group are given below:-

	NO. CASES	OXYGEN SATURATION % VEIN	OXYGEN SATURATION % ARTS.	HB. GMS.%
I. NORMAL PREGNANCY				
No cord obstruction				
A. Less than 39 weeks	26	72.7	38.2	17.0
B. 39 - 41 weeks	60	64.4	31.2	17.4
C. Over 41 weeks	26	53.9	20.2	17.8
II. NORMAL PREGNANCY				
Cord round neck (39 - 41)	6	56.1	11.7	17.3
Delayed Delivery (39 - 41)	3	48.5	14.8	18.9
III. NORMAL TWIN PREGNANCY				
Breech (32 - 38)	3	37.9	18.1	17.6
Vertex (32 - 38)	4	41.0	21.7	16.8
Total	7	39,7	20.1	17.1
IV. TOXAEMIC PREGNANCY				
A. Less than 39 weeks	9	51.2	21.2	17.6

		NO. CASES	OXYGEN SATURATION % VEIN	OXYGEN SATURATION % ARTS.	HB. GMS.%
B.	39 - 41 weeks	23	50.6	18.7	17.9
	(1) Generalised Oedema	4	54.3	21.0	17.1
	(2) Mild	5	53.0	16.4	17.7
	(3) Moderate	10	51.0	19.5	18.1
	(4) Severe	4	42.8	17.0	18.4
	Duration less than 3 weeks	6	56.5	20.6	17.5
	Duration 3 weeks or more	17	48.5	18.0	18.0
C.	Over 41 weeks	6	45.0	14.6	18.4
V.	PREGNANCY WITH HYPERTENSION	8	71.1	34.8	18.0
VI.	FORCEPS DELIVERIES				
	Normal Pregnancy	5	55.8	15.8	18.5
	Toxaemic Pregnancy	9	43.9	15.7	17.6
VII.	CAESAREAN SECTION				
	Normal Pregnancy	13	42.7	19.1	16.3
	Toxaemic Pregnancy	10	(13.9-) (58.6)	(4.3) (25.4)	(14.2-) (19.9)
	Diabetic Pregnancy	13	37.1	12.7	16.7

DISCUSSION

A. NORMAL PREGNANCY

Although the levels of the oxygen content in the umbilical cord blood immediately after an uncomplicated vaginal delivery may not give a true indication of the conditions "in utero", they are more likely to do so than those in which additional factors such as cord obstruction, delay, instrumental and operative procedures further influence the conditions.

It has been shown that in normal cases at term the oxygen content of the blood reaching the foetus shows a wide range of results (10.65 - 18.46 vols.%) with an average result of 15.06 vols.% (60 cases) giving an oxygen saturation of 64.4% (55.7 - 74.9). The average saturation of 26 cases delivered before the 39th week was significantly higher (72.7%) while that of 26 cases delivered after 41 weeks was significantly lower (53.9%). Parallel changes occurred in the levels of oxygen content.

It may therefore be suggested that as pregnancy matures the blood leaving the placenta is progressively less well oxygenated. This was suggested by Walker[33] (1953), studying human cord blood. Barcroft et al[34] (1940) showed a similar drop in the levels of oxygen content and saturation in the sheep foetus with

maturing pregnancy and Barcroft and Young[56](1943) showed a progressive deterioration in the oxygen levels of blood from the cranial venous sinuses of foetal rabbits as postmaturity continued.

In all groups and the great majority of individual cases it was shown that there was a fairly constant difference of 30 - 35% saturation between the vein and arteries - foetal requirements apparently being fairly constant under normal conditions. The actual difference in the oxygen content in the three main groups also being constant (7.80, 7.75 and 7.96 vols.%) - slightly higher in the postmature group. Although the average baby weight increased with maturity, there is no marked increase in the arterio-venous difference. This may be explained by an increase in the circulation rate with the maturity and size of the foetus. Unfortunately this is impossible to measure "in utero" so we must accept that foetal requirements are apparently 30 - 35% saturation. Foetal distress has been shown to be associated with a saturation of 30% or less in the umbilical vein.

Let us consider why the levels become lower with increasing maturity. The oxygen levels in the umbilical vein are dependent on a number of factors:-

(1) the oxygen content of maternal blood.

(2) the blood supply to the uterus and placenta.

(3) the oxygen transfer in the placenta.

(4) the oxygen combining power of the foetal haemoglobin.

(5) free circulation in the cord.

In all groups (1) presumably remained reasonably constant, although Eastman[25](1936) showed the maternal arterial saturation in labour to be slightly lower (95%) than the normal 98%. With regard to (5) - as far as possible all cases of potential cord obstruction were excluded from the main groups. The few cases studied showed a lower level of oxygen saturation in the vein and a much lower level in the arteries as was shown by Clemetson[32](1953) - the increased difference being due to a slowing of the circulation rate in the cord.

With regard to the haemoglobin, it is generally agreed that foetal haemoglobin has a much greater affinity for oxygen than adult type. Although it has been shown by Walker[57](1955) and others that there is an increasing amount of adult haemoglobin present as pregnancy matures, which might lead to a diminution of the affinity for oxygen, Darling et al[58](1941) showed that foetal oxygen dissociation curves at 5 months, 7 months and term were very similar. As the

haemoglobin levels showed only a slight and not highly significant rise with maturity we may assume that this factor remained reasonably constant in all groups.

The main difference must therefore lie in the blood supply to the uterus and placenta and the oxygen transfer. The blood supply to the uterus, shown by Assali et al[59](1953) to be on an average 15 ml./100 mgms./minute in the last few weeks of pregnancy, may become impaired as pregnancy matures to term and thereafter. Hamilton[60](1949) showed a diminished cardiac output in the last few weeks of pregnancy. The uterus and foetus are increasing in size and, apart from increasing requirements, mechanical factors such as increased intra-uterine and intra-abdominal pressure may well reduce the uterine blood supply. It is also possible that in cases in which the pregnancy has continued far beyond the expected date of delivery some "emotional tension" may be present, which, at least theoretically, might cause some spasm of the spiral arterioles due to some psycho-somatic factor affecting the autonomic nervous system.

Bartholomew[61](1951) suggested that Braxton Hicks contractions towards term might further impair the placental blood supply. Woodbury et al[62](1938) showed that the effective maternal arterial pressure to the

placenta was diminished, sometimes to zero, during uterine contractions. Smith and Smith[63](1948) suggested that the fall in oestrogens and progesterone towards term might reduce the vascularity of the uterus which they normally maintain.

With regard to the twin pregnancies studied, the low levels are probably associated with the increased intra-uterine pressure mentioned above.

It has been shown that the placenta itself receives its nourishment from the maternal blood supply (Van Wagenen[64] 1943, Hellman[65] 1947) and increasing placental ischaemia with maturity has been shown by many investigators:- (a) degenerative histological changes - Tenney[2] 1936, [3]1940; Wang and Hellman[66] 1943 (associated with a lower oxygen consumption); Bartholomew[61] 1951; Hughes et al[4] 1954; Clifford[44] 1954; (b) functional changes have been shown by Page[5] 1939, [6]1946; Smiths[67] 1941; Thompson and Tickner[68] 1949; Hughes et al[4] 1954 etc.

Bartholomew[61](1951) has suggested that some spasm of the sphincters of the collecting veins in the foetal circulation (shown by Spanner[69]) - in response to some oxytocic factor released from an inhibitor (such as oestrogen or pitocinase) may be the initial factor in the congestion and degeneration of the villi. The

actual precipitating cause of the degeneration (whether it be in the maternal or foetal circulation) is of little importance compared with the fact that it does occur and does interfere mechanically with placental transfer. The degeneration has been shown to include congestion of the villi causing a narrowing of the intervillous channels (also probably caused by increased intra-uterine pressure) and a hyaline and fibrin formation between the maternal and foetal blood which would mechanically reduce permeability. Flexner et al[70] (1948) showed that the transfer of radio-active sodium across the placenta was appreciably diminished from about 36 weeks to term - again showing the effects of progressive degeneration.

This impairment of transfer would account for the higher levels of carbon dioxide found as maturity increased.

Should the blood supply become markedly impaired placental infarction occurs and thereby reduces the transfer even further - a higher incidence of infarction is shown in post-mature placentae (Gibberd[71] 1952, Clifford[44] 1954). Clifford has also pointed out the progressive yellow staining of the foetus and its loss of nutrition in pregnancies of excessive duration - due to failing placental function.

As pregnancy is prolonged a progressive impairment

of the oxygenation of the foetal cord blood may be expected as the effective blood flow and permeability of the placenta are being diminished. It has been shown that the foetal requirements appear to be 30 - 35% oxygen saturation therefore as that figure is approached in the level of saturation in the vein only the necessary requirements are reaching the foetus. At term there is 100% reserve but as pregnancy is continued over 41 weeks this reserve is progressively diminished (N.B. the case delivered by Caesarean section 44 weeks - with marked yellow staining - vein 37.4%; one spontaneous vertex delivery 42 weeks - vein 37.3%, showing gross placental infarction). In 1937, Snyder and Rosenfeld[72] showed that postmature rabbits made respiratory movements "in utero" - also showing that foetal requirements were not being adequately provided for.

In the majority of cases the lowered oxygen reserve may be of little importance but should there be any sudden placental insufficiency or cord obstruction "in utero" or should the patient have a difficult labour, as is more common in postmature cases (Rathbun[40] 1943; Stewart[73] 1952; Clayton[74] 1953; Walker[45] 1954, etc.) and require an instrumental or operative delivery, these factors will certainly expose the foetus to grave

dangers as all may cause a further lowering of the oxygen saturation in the vein. This probably accounts for the increased number of unexplained foetal deaths (Gibberd[71]; McKiddie[41]; Rathbun[40]; Walker[45]; etc.) and the high incidence of foetal distress (Clayton[74]; Walker[45];) and of anoxia as the sole cause of death being found in postmature cases. All increase progressively with further prolongation of pregnancy.

The difficulty lies in determining clinically when the pregnancy becomes postmature - as it is common knowledge that the duration of pregnancy is variable, although the average is forty weeks, and the date of the last menstrual period is often in doubt. Unless the obstetrician is convinced that the pregnancy is not over 42 weeks it would seem advisable to try to induce labour at that time, for, although there was marked overlapping of the results, the average oxygen saturation in the umbilical vein in cases of that duration was already 11% less than those at term (oxygen reserve reduced by one-third) and induction per se has not been shown to have any effect on the oxygen levels at birth.

B. PREGNANCY COMPLICATED BY PRE-ECLAMPSIA

In cases complicated by pre-eclampsia the average oxygen saturation of the umbilical vein at term was shown to be significantly lower (50.6%) than that in normal pregnancies (64.4%). The level was shown to be progressively lower (54.3%, 53.0%, 51.0%, 42.8%) with the increasing severity and duration of the toxaemia (under 3 weeks - 56.5%, over three weeks 48.5%).

In almost all the severe cases of long duration macroscopic placental infarction was present - associated with the lowest levels of oxygen saturation recorded.

In cases of pre-eclampsia, although mild, where pregnancy was prolonged over 41 weeks, all but one placenta showed infarction and the average arterial oxygen saturation was 45% - nearly 20% lower than the normal term value.

In premature severe cases the average oxygen saturation was again 45%. All were of at least three weeks duration and all showed placental infarction. Even when mild and severe premature cases were grouped together, the average oxygen saturation was 21.5% lower than that of normal premature pregnancies.

The arterio-venous difference in saturation was on an average 30 - 33% except in the severe cases

where it was slightly lower (premature 26.8%; term 25.8%). In these groups the average baby weights were lower than normal (premature 4 lb. 7 ozs., term 6 lb. 7 ozs.) The saturation in the umbilical arteries was more or less parallel to that in the vein.

The haemoglobin levels were significantly higher than normal in all but the mild cases and showed a further increase with the duration of the pre-eclampsia.

The oxygen contents of the vein and arteries were significantly lower throughout and the lower saturation was due very largely to this rather than to the increased oxygen capacity of the blood, associated with the increased haemoglobin.

There was a rise in the level of carbon dioxide in both vessels - the difference (6 vols.%) being similar to that found in normal pregnancy.

As with normal pregnancy a number of factors determine the oxygen levels in the cord at birth.

In this instance, only the lack of cord obstruction may be excluded from consideration.

With regard to the maternal circulation, several changes have been shown to be associated with pre-eclampsia. Hamilton[60] (1949) showed that the cardiac output was increased in pre-eclampsia, although Werko[75] (1950) did not find this. Dieckmann[76] (1952) has

shown an increased relative blood viscosity in pre-eclampsia (not shown by Hamilton[77], 1950) and a variable degree of haemoconcentration associated with a diminished blood volume. The last was also shown by White [78] (1949) in 41 cases of pre-eclampsia studied. This haemoconcentration will tend to produce an impaired blood flow to all organs even with the increased cardiac output and it has been shown that it also causes interference with the normal oxygenation of the blood in the lungs - concentrated blood never being fully saturated by lung perfusion (Dieckmann).

Dieckmann[76] has also suggested that a higher proportion of inactive haemoglobin may be present in patients suffering from pre-eclampsia - particularly when heavily sedated. The effect of all these changes might be some impairment of the oxygen supply of the blood reaching the uterus.

A significant increase was shown in the level of haemoglobin in the cord blood. As was suggested by Walker[33] (1953) this is probably a response by the foetus to a prolonged period of relative anoxia. Very few of the cases studied showed dangerous levels of oxygen saturation in the umbilical vein but almost all, particularly those of long duration, had much lower levels than normal and probably more difficulty

was encountered in getting this quantity of oxygen in the placenta. Although there is an increase in total haemoglobin, Walker[57](1955) showed that this increase was almost entirely foetal type so the oxygen dissociation curve is not materially altered - except by becoming more favourable to the foetus.

The blood supply to the uterus in pre-eclampsia was, until recently, largely a matter of conjecture. Radio-active sodium has been used by Browne and Veall[79](1953) to determine the placental blood flow, which was shown to be reduced to about one third of the normal (600 ml./min. at 38 - 40 weeks) in cases of pre-eclampsia. In 1955, Morris et al[80] used a similar method of determining uterine blood flow, with no special reference to the placental site, and showed it also to be considerably reduced ($\frac{1}{2}$ normal in mild pre-eclampsia and $\frac{1}{4}$ normal in severe cases). The latter further showed a two-or three-fold increase in the blood flow in cases treated with hypotensive drugs. The uterine blood flow was also found to be diminished in twin pregnancies - probably due to mechanical factors. As was mentioned earlier the increased cardiac output does not apparently cause an increased blood supply to the uterus. Kellar[81](1949) noted that in several cases of pre-eclampsia the uterine

venous blood appeared to be better oxygenated than normal - suggesting some failure of utilisation of oxygen.

Apart from factors affecting the placental blood supply in normal pregnancy, there is additional interference. The general arteriolar spasm of the maternal circulation, associated with pre-eclampsia (Eastman[82], 1937; Van Bouwdijk Bastiaanse[12], 1954, etc.) will presumably affect the uterine vessels. These have been shown by Beker[11](1948) to be smaller than normal in eclamptic patients, and particularly primigravidae. Theobald[83](1953), by denervation of the internal iliac arteries in three hypertensive patients who had had stillbirths, produced three living children by increasing the uterine blood supply. He also noted that the internal iliac vessels were reduced in size in two cases of pre-eclampsia.

Since 1914, when Young[1] suggested interference with the placental blood supply as a cause of the placental infarction associated with pre-eclampsia, the theory that placental ischaemia and pre-eclampsia go hand in glove has become more and more widely accepted whether as Page[8](1948) said "relative maternal ischaemia of the placenta is the proximate or the precipitating cause of pre-eclampsia."

A large number of investigations relating the two have been carried out and only a brief outline of some can be given here.

Firstly Page (1939[5], 1948[8]) re-emphasised the association between the incidence of toxaemia and the possible causes of placental ischaemia. An impaired blood supply due to increased (a) intra-abdominal and (b) intra-uterine pressure was comparable with the increased incidence of pre-eclampsia in (a) primigravidae and obese patients (b) hydatidiform mole, multiple pregnancy, hydrammios, towards term. Previous vascular disease and severe anaemias (e.g. hookworm) were associated with the higher incidence in essential hypertension and diabetes and in cases of marked maternal anaemia.

Ogden et al[9](1940) and Van Bouwdijk Bastiaanse and Mastboom[84](1949) produced hypertension in pregnant dogs by causing artificial uterine ischaemia. As a similar effect was not shown in the non pregnant animals it was concluded that the result was due to placental, rather than uterine, ischaemia.

Parviainen et al[85](1951) showed increased uterine tone in pre-eclampsia and Bartholomew[61](1951) stressed the incidence of eclampsia in labour where, as was mentioned earlier, Woodbury et al[62](1938) showed that

the effective maternal arterial pressure to the placenta was considerably reduced during contractions. Conversely Smith [86](1947) and Van Bouwdijk Bastiaanse[12] (1954) associated the lower incidence of pre-eclampsia during famine years with an improved uterine blood supply due to loss of tone of the muscles of the uterus and blood vessels.

Smith and Smith[63](1948) suggested an impaired blood supply due to the fall in oestrogens which normally increase uterine vascularity - producing a vicious circle by causing further hormonal disturbance. They advocated the administration of oestrogens as a method of treatment but this has not been shown satisfactory.

Degenerative histological changes, as found in normal mature placentae, occur to a much greater and often diagnostic degree in pre-eclampsia - the extent depending on the severity, time of onset and duration of the disease. This has been shown by many authors including Tenney (1936[2], 1940[3]); Wislocki and Dempsey[87](1946) who also showed associated histochemical changes of advanced ageing; Bartholomew (1938[88], 1947[89]) and Hughes[4](1954). Page[8](1948) showed that this degeneration was associated with a diminished oxygen consumption of the placental tissue

in severe cases of pre-eclampsia. These degenerative changes help to account for the high incidence of macroscopic infarction and premature placental separation.

Functional changes have been widely studied and in many cases the disturbances shown have been associated with placental ischaemia, e.g. disturbed pitocinase levels (Page[6], 1946), histaminase (Ahlmark[90] 1944; Kapeller-Adler[91], 1951), angiotonase (Page[7], 1947).

Hormonal imbalance in pre-eclampsia has also been widely investigated e.g. Smith and Smith[92], 1943; Tobian[93], 1949; Loraine and Matthew, 1950[94], 1953[95]; Hughes et al[4], 1954; Venning et al[96], 1954, etc., etc. - many authors stressing the effect of failing placental physiology associated with the disease.

In 1949, Thompson and Tickner[68] showed that a monoamine oxidase capable of inactivating vaso-constricting amines was present in placental tissue and that "in vitro" its activity was proportional to the oxygen tension of its medium - hence pointing out the connection between placental ischaemia and pre-eclampsia.

In 1951, Green et al[13] showed a rise in the average pentose and phosphorylated pentose in the plasma of women suffering from pre-eclampsia - this they attributed

to the effects of uterine and placental ischaemia.

Although no definite conclusion has been reached as to whether or not the placental ischaemia precedes the pre-eclampsia, it is sufficiently evident that in pre-eclampsia the blood supply to the placenta is impaired. This blood supply must serve the needs of the placental tissue itself apart from the first consideration of foetal requirements therefore, as the latter increase, and the blood supply becomes increasingly impaired as might be expected, the placenta will become progressively more anoxic and degenerated. The improvement in pre-eclampsia following the death of the foetus may be due to an increased oxygen supply to the placenta with the removal of foetal requirements as was suggested by Beker[11](1948).

This ischaemia and degeneration of the placenta - even without the appearance of gross infarcts - will materially alter the oxygen transfer. The associated congestion of the villi (Tenney[2,3]; Falkiner[97], 1944; Hughes[4]) reduces the intervillous channels and the hyaline fibrin deposition further impairs the permeability of the placenta.

Placental transfer was shown to be impaired in pre-eclampsia by Flexner[70](1948) and Cox and Chalmers[98] (1953) using radio-active sodium, and in 1954 by

Clemetson and Churchman[99] who showed an impairment of the transfer of amino acids to the foetus in pre-eclampsia.

We have therefore an impaired supply of probably poorly oxygenated blood reaching a placenta where the efficiency of transfer is becoming progressively impaired. This accounts for the lowering of the oxygen saturation in the umbilical vein in cases of pre-eclampsia and explains why it is more marked in severe cases and cases of long duration. The higher levels of carbon dioxide are probably also due to the impaired blood supply and permeability of the placenta as the difference between the levels in the vein and arteries is similar to that in normal pregnancy.

This lowering of the available oxygen, associated with the generally impaired nourishment of the foetus - due to progressive placental insufficiency - will account for the increased mortality, particularly intra-uterine death, found in pre-eclampsia. (Ludlow[14], 1933; Wellen[18], 1940; Drillien[16], 1947; Brash[21], 1949; etc.). Even at spontaneous vaginal delivery the oxygen saturation was much lower than normal - and with the increased incidence of instrumental and operative deliveries, carried out for the sake of the mother, the foetus is often exposed to very serious

danger of anoxia. There is also the additional danger of premature separation of the ischaemic placenta which becomes more imminent with progressive degeneration.

As in the normal cases the foetal requirements again appeared to be about 30% except in severe cases where it was slightly lower, probably due to the fact that the smaller undernourished infants have lower basal requirements. This danger level of 30% saturation in the vein has been shown to occur in several cases by all methods of delivery. It may be purely momentary but does give an indication that further interference with the oxygen supply might prove fatal.

In view of the preceding results which have tended to show that the impairment of oxygen supply is progressive - and appreciable in even mild cases, the question of the best procedure to adopt arises. With regard to severe toxaemia in early cases, the foetus will still remain a purely secondary consideration as premature induction will produce a very small infant with little likelihood of survival. On the other hand, if the foetus is sufficiently mature to stand a reasonable chance of survival it would appear to be advantageous to deliver it before it runs an even greater risk of intra-uterine anoxia - preferably

without exposing it to the additional dangers of an instrumental or operative procedure. There certainly appears to be no advantage in allowing even mild cases of pre-eclampsia to proceed beyond 41 weeks gestation as it has been shown that the oxygen reserve is reduced to an average 14.6% by the 42nd week - and in some cases even lower.

C. PREGNANCY COMPLICATED BY HYPERTENSION

This group of 8 cases is too small to draw any definite conclusions and merits further investigation. Although the average oxygen content and saturation in the cord vessels appeared to be higher than that in normal mature pregnancy this may not give a true picture as almost all were very mild cases of hypertension - the maximum blood pressure recorded at delivery being $\frac{150}{105}$.

The maternal circulatory changes associated with pre-eclampsia such as increased cardiac output and diminished blood volume have not been shown to occur in cases complicated by hypertension alone (Kellar[100], 1950; White[78], 1949). Vascular changes are possibly present but in mild cases the extent of these is probably minimal and insufficient to affect the placental blood supply. Two of the cases studied had a history of hypertension but no pre-eclampsia in previous pregnancies which, in view of the association of placental ischaemia and pre-eclampsia, tends to show that the blood flow to the uterus and placenta was adequate for both foetal and placental needs.

Wang and Hellman[66](1943) showed that the placentae from cases of chronic hypertension were essentially the same as those from normal pregnancy. The oxygen

consumption of the tissue was not materially altered, nor was the histology. Tenney[3](1940) also showed no degenerative change associated with hypertension unless pre-eclampsia was superimposed.

Until further results are obtained, particularly from cases showing a more marked elevation of blood pressure, it seems reasonable to suggest that there may be no impairment of the oxygenation of the foetus in cases of mild hypertension without the added complication of superimposed toxaemia, which is of course more liable to develop in cases of pre-existing hypertension as was mentioned earlier.

CONCLUSIONS

1. In 60 cases of normal pregnancy delivered spontaneously at term the average oxygen saturation in the umbilical cord was shown to be

64.4 ± 4.53% (vein)

31.2 ± 4.53% (arteries)

2. In 26 cases delivered before 39 weeks the average oxygen saturation in both vessels was significantly higher

72.7 ± 4.62% (vein)

38.2 ± 4.29% (arteries)

3. In 26 cases delivered after 41 weeks the average oxygen saturation in both vessels was significantly lower

53.9 ± 5.63% (vein)

20.2 ± 6.13% (arteries)

There was probably a progressive danger of anoxia as pregnancy was unduly prolonged which was enhanced by the increase in instrumental and operative deliveries. There is an indication for terminating pregnancies no later than the 42nd week.

4. In 38 cases of pre-eclampsia the oxygen saturation

was considerably impaired in most instances. This was increased with the severity and duration of the toxaemia. It is unwise to allow any pregnancy complicated by even a mild degree of pre-eclampsia to proceed beyond term in view of the increasing danger of foetal anoxia "in utero" and during delivery.

5. In 8 cases complicated by mild hypertension the oxygen saturation was

71.1 ± 3.41% (vein)

34.8 ± 3.92% (arteries)

There is no indication of impaired oxygenation and placental insufficiency in these few cases, but the group requires further study.

6. Instrumental and operative deliveries or some factor associated with them are liable to expose the foetus, even momentarily, to extremely low oxygen levels.

7. At birth, after surgical induction of labour, the oxygen levels in the cord were similar to those obtained in cases where labour was not induced.

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TABLE A

NORMAL PREGNANCY - 35 and 36 WEEKS

Duration of Pregnancy	Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight	Placenta weight	Coefft. of Oxygen Utiln.
		Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb. ozs	lb. ozs	
35	181	17.17	16.2	79.1	9.64	16.4	43.9	33.22	37.66	4 1½	1 1	0.44
36	4	18.10	18.1	74.6	9.94	18.2	40.8	40.22	47.09	5 14¼	1 8	0.45
	29	17.73	18.3	72.3	9.08	17.6	38.5	43.35	48.74	6 2¼	1 8	0.49
	81	15.83	15.3	77.2	9.45	14.9	47.3	38.93	46.87	6 7½	1 2	0.40
	186	15.63	16.7	69.9	7.78	16.9	34.4	41.75	47.25	5 7¾	1 2	0.50
AVERAGE		16.82	17.1	73.5	9.06	16.9	40.3	41.06	47.49	6 0	1 5	0.46
RANGE		15.63	15.3	69.9	7.78	14.9	34.4	38.93	46.87	5 7¾	1 2	0.40
		18.10	18.3	77.2	9.94	18.2	47.3	43.35	48.74	6 7½	1 8	0.50

TABLE B

NORMAL PREGNANCY - 37 WEEKS.

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight		Placenta weight		Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb.	ozs	lb.	ozs	
55	18.84	18.2	77.3	9.59	18.1	39.5	34.27	42.57	6	8½	1	5	0.49
73	16.59	16.6	74.6	9.10	16.9	40.2	38.82	43.27	7	7¼	1	6	0.45
85	16.82	15.9	78.9	8.30	16.2	38.2	39.79	47.52	7	0¼	1	13	0.51
116	18.29	16.6	82.2	7.41	16.9	32.7	43.14	50.38	5	13	1	2	0.59
122	14.52	15.2	71.3	9.51	15.7	45.2	38.43	43.12	5	12	1	11	0.35
148	16.05	15.7	76.3	9.21	15.8	43.5	34.98	39.28	6	9	1	6	0.43
159	16.78	17.7	70.7	10.68	18.5	43.1	37.40	44.10	6	10¼	1	4	0.36
161	16.12	15.9	75.7	8.00	15.7	38.0	38.00	42.99	6	0	0	15	0.50
212	16.18	17.5	69.0	8.59	17.8	36.0	42.35	47.09	5	6½	1	0½	0.47
224	17.30	17.9	72.1	9.21	17.9	38.4	41.27	46.11	5	10½	1	3	0.47
239	15.24	15.9	71.5	7.43	15.7	35.2	38.61	42.51	5	12¼	1	2	0.51
AVERAGE (11)	16.61	16.6	74.5	8.82	16.8	39.1	38.82	44.45	6	3¾	1	4¾	0.47
RANGE	14.52	15.2	69.0	7.41	15.7	32.7	34.27	39.28	5	6½	0	15	0.35
	18.84	18.2	82.2	10.68	18.5	45.2	43.14	50.38	7	7¼	1	13	0.59

TABLE C

NORMAL PREGNANCY - 38 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight	Placenta weight	Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb. ozs	lb. ozs	
13	16.56	19.2	64.4	8.35	19.6	31.8	40.82	44.94	7 5¾	1 0	0.50
32	16.75	16.7	74.9	7.78	16.6	35.0	36.83	40.83	5 14¼	1 2	0.54
38	13.96	16.7	62.4	7.27	16.7	32.5	40.75	45.69	6 0½	1 1	0.48
74	16.09	17.1	70.2	8.62	17.2	37.4	40.52	46.67	7 2	1 3	0.54
80	16.27	16.2	74.9	9.70	16.6	43.6	36.42	45.85	6 6¾	1 4	0.46
95	16.56	18.5	66.8	8.12	18.9	32.1	39.91	42.65	8 0	1 10	0.51
133	16.77	17.4	71.9	9.08	18.2	37.2	40.71	46.63	9 4	1 6	0.46
177	17.35	17.9	72.3	8.64	18.1	35.6	37.78	45.06	5 8½	0 15	0.50
194	15.09	16.9	66.6	7.74	16.9	34.2	40.61	47.91	6 13	1 3	0.49
199	17.80	18.4	72.2	9.34	18.1	38.5	42.84	47.51	6 2½	1 4	0.48
AVERAGE (10)	16.32	17.5	69.7	8.46	17.7	35.8	39.72	45.37	6 13¾	1 3¼	0.50
RANGE	13.96	16.2	62.4	7.27	16.6	31.8	36.42	40.83	5 8½	0 15	0.46
	17.80	19.2	74.9	9.70	19.6	43.6	42.84	47.91	9 4	1 10	0.54

TABLE D

NORMAL PREGNANCY - 39 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight		Placenta weight		Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb.	ozs	lb.	ozs	
36	12.49	16.4	56.8	5.88	16.0	27.4	40.39	47.44	5	10¾	1	7	0.53
65	15.81	17.6	67.2	7.59	17.8	31.8	36.52	44.28	6	10¼	1	4	0.52
82	15.94	18.3	65.0	7.63	18.1	31.5	36.21	42.81	7.	1	1	4	0.52
94	14.66	16.1	68.0	6.97	16.0	32.5	47.09	50.63	7	1	1	8	0.52
107	18.46	20.1	68.5	8.04	19.8	30.3	41.00	52.15	7	12½	1	8	0.56
124	16.28	18.6	65.3	8.74	18.3	35.6	36.62	42.71	6	7	1	2	0.46
126	16.45	18.7	65.6	7.34	18.5	29.6	39.23	46.45	6	14	1	7	0.55
132	13.56	16.8	60.2	6.39	17.2	27.7	42.16	48.20	6	13¼	1	6	0.53
142	17.91	18.9	70.7	8.41	19.6	32.0	42.16	49.43	7	0	1	8	0.53
149	14.76	18.2	60.5	6.73	18.7	26.9	47.27	52.71	6	10¼	1	5	0.54
160	14.09	15.6	67.4	7.61	15.6	36.4	37.29	43.04	7	3¼	1	5	0.46
203	13.40	15.6	64.1	7.24	16.0	33.8	44.68	49.50	7	3½	1	9	0.46
221	15.54	18.8	61.7	6.67	18.9	26.3	37.43	42.56	7	7	1	3	0.57
AVERAGE/													

TABLE D Continued NORMAL PREGNANCY - 39 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight	Placenta weight	Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb. ozs	lb. ozs	
AVERAGE (13)	15.33	17.7	64.7	7.33	17.7	30.9	40.62	47.07	6 $14\frac{1}{2}$	1 $5\frac{3}{4}$	0.52
RANGE	12.49	15.6	56.8	5.88	15.6	26.3	36.21	42.56	5 $10\frac{3}{4}$	1 2	0.46
	18.46	20.1	70.7	8.74	19.8	36.4	47.27	52.71	7 $12\frac{1}{2}$	1 9	0.57

TABLE E

NORMAL PREGNANCY - 40 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight	Placenta weight	Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb. ozs	lb. ozs	
6	14.76	17.1	64.4	7.42	17.3	32.0	39.24	46.84	6 14½	1 6	0.50
9	10.65	13.7	58.0	4.78	13.8	25.8	47.85	51.50	6 12¼	1 7	0.55
28	13.92	17.5	59.4	6.37	13.1	26.3	40.23	45.88	6 5½	1 5	0.54
39	13.39	14.5	68.9	6.65	16.1	30.8	42.99	46.91	7 7¾	1 8	0.50
40	13.24	16.4	60.2	4.62	16.7	20.6	42.14	47.21	7 5	1 9	0.47
41	13.37	15.1	66.1	6.54	15.2	32.1	44.04	48.93	7 3½	1 10	0.51
46	14.48	17.9	60.4	7.17	17.8	30.1	37.16	45.81	6 15	1 4	0.50
49	16.11	19.8	60.7	8.50	19.9	31.9	38.68	44.74	5 14	0 15	0.47
60	13.72	17.8	57.5	6.29	18.4	25.5	40.13	45.10	6 12½	1 0	0.54
77	17.27	18.0	71.6	9.92	18.5	40.0	37.54	42.89	5 10½	0 15	0.43
84	17.30	17.9	72.1	8.20	18.2	33.6	43.75	47.43	7 2¾	1 9	0.53
86	15.66	19.2	60.9	6.91	19.2	26.9	42.72	49.82	7 4	1 2	0.56
92	14.84	16.7	66.3	7.72	16.5	34.9	39.94	44.86	7 15½	1 10	0.48

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TABLE E Continued — NORMAL PREGNANCY - 40 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight		Placenta weight		Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb.	ozs	lb.	ozs	
97	13.57	15.6	64.9	6.20	16.0	28.9	40.88	48.97	9	7	1	11	0.48
99	16.82	19.2	65.4	8.30	19.2	32.3	39.79	46.52	7	11½	1	9	0.51
106	13.89	18.6	55.7	6.03	18.6	24.2	37.86	46.38	7	13¾	1	9	0.51
119	15.71	17.4	67.4	6.40	17.4	27.4	41.60	46.90	7	11	1	7	0.59
128	17.93	19.2	69.7	9.37	18.9	37.0	38.51	45.53	9	7¾	1	11	0.48
129	13.06	16.9	57.7	6.13	16.5	27.7	43.82	50.42	7	6½	1	11	0.53
139	16.03	16.7	71.6	6.82	16.3	31.2	39.70	47.94	8	2¼	1	11	0.57
140	16.32	18.6	65.5	8.77	18.5	35.4	40.25	48.27	8	4	1	11	0.46
143	12.52	15.6	59.9	7.62	15.7	36.2	41.44	46.24	8	3	1	8	0.39
150	14.76	16.2	68.0	6.49	16.0	30.3	46.53	51.69	7	4¼	1	2	0.56
157	13.28	17.2	57.6	8.31	18.1	34.3	39.49	45.88	7	7	1	9	0.37
201	14.93	17.2	64.8	6.94	16.7	31.0	43.43	48.26	7	2	1	5	0.54
206	14.05	16.8	62.4	7.59	16.9	33.5	41.81	44.99	8	12¼	1	8	0.46

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TABLE E Continued — NORMAL PREGNANCY - 40 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight		Placenta weight		Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb.	ozs	lb.	ozs	
208	15.06	17.1	65.7	7.48	16.7	33.4	42.69	47.27	8	2½	1	5	0.50
210	14.71	17.6	62.4	7.39	17.4	31.7	41.46	47.88	7	3	1	3	0.50
230	13.16	16.3	60.3	6.97	16.5	31.5	43.13	48.30	7	7¼	1	3	0.47
233	14.77	18.3	60.2	8.26	18.5	33.3	43.67	47.61	7	9	1	8	0.44
AVERAGE (30)	14.64	17.2	63.5	7.21	17.3	31.0	41.42	47.23	7	8	1	6½	0.50
RANGE	10.65	13.7	55.7	4.62	13.8	20.6	37.16	42.89	5	10½	0	15	0.37
	17.93	19.8	72.1	9.92	19.9	40.0	47.85	51.69	9	7¾	1	11	0.59

TABLE F

NORMAL PREGNANCY - 41 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight		Placenta weight		Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb.	ozs	lb.	ozs	
7	17.75	20.3	65.3	8.24	20.2	30.4	36.40	44.73	7	11½	1	8	0.54
20	17.69	18.4	71.7	8.19	18.6	32.9	37.04	43.69	7	10¾	1	4	0.54
34	16.11	19.0	63.3	7.98	19.0	31.3	40.56	48.32	5	7½	1	7	0.50
44	18.16	19.1	71.0	9.17	19.4	35.3	37.04	41.70	7	6	1	6	0.50
101	15.72	18.1	64.8	6.71	18.5	27.1	40.36	48.96	8	10¼	1	12	0.57
104	17.26	17.2	74.9	10.55	17.2	45.8	40.06	46.28	6	12¼	1	6	0.39
105	18.27	20.6	66.2	5.36	19.7	20.3	40.25	48.57	6	13¾	1	9	0.71
123	14.09	16.6	63.3	5.16	16.4	23.5	42.04	47.43	7	12½	1	5	0.63
125	15.95	17.0	70.0	8.77	17.0	38.5	36.51	40.67	7	9½	1	7	0.45
135	13.19	17.6	55.9	6.36	17.8	26.7	42.40	45.29	7	12½	1	7	0.52
146	13.78	17.0	60.5	7.53	17.5	32.1	40.05	45.28	9	7	1	11	0.45
172	14.16	15.5	68.2	7.34	14.7	37.3	43.12	49.55	8	2¾	1	4	0.48
202	13.49	16.7	60.3	7.48	17.0	32.8	45.51	49.52	8	2½	1	7	0.45

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TABLE F Continued — NORMAL PREGNANCY - 41 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight	Placenta weight	Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb. ozs	lb. ozs	
207	15.00	17.0	65.8	8.24	17.2	35.8	40.59	43.97	7 13	1 12	0.45
211	14.99	16.7	67.0	6.92	16.8	30.7	44.52	49.61	7 3	1 5¼	0.54
218	15.38	17.4	66.0	6.24	17.0	27.4	38.03	46.66	8 1	1 10	0.59
231	13.75	16.3	63.0	7.23	16.4	32.9	42.86	46.34	7 6	1 8	0.47
AVERAGE (17)	15.57	17.7	65.7	7.50	17.7	31.8	40.43	46.27	7 9½	1 7½	0.52
RANGE	13.19	15.5	55.9	5.16	14.7	20.3	36.40	40.67	5 7½	1 4	0.39
	18.27	20.6	74.9	10.55	20.2	45.8	45.51	49.61	9 7	1 12	0.71

TABLE G

NORMAL PREGNANCY - 42 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight		Placenta weight		Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb.	ozs	lb.	ozs	
2	14.84	21.0	52.7	4.21	22.2	14.2	41.02	47.41	7	14¾	1	3	0.76
19	14.80	19.0	58.1	7.34	19.9	27.5	39.85	49.62	8	4¼	1	4	0.50
83	15.55	19.9	58.3	6.52	20.1	24.2	37.09	47.84	8	0	1	6	0.58
90	10.14	20.3	37.3	3.12	20.1	11.6	34.89	44.83	6	4	1	2	0.69
98	12.90	19.0	50.7	3.39	18.9	13.4	38.68	50.33	7.	8	1	4	0.74
112	10.47	14.6	53.5	3.92	14.2	20.6	47.26	51.50	9	6	2	5	0.63
144	14.00	16.1	64.9	7.89	16.7	35.3	42.53	45.71	8	4	1	9	0.44
153	9.59	13.4	53.4	1.84	12.6	10.9	43.79	52.22	7	14¼	1	11	0.81
156	11.30	15.8	53.4	4.46	15.6	21.3	41.68	44.18	8	11½	1	10	0.61
164	12.64	18.9	49.9	4.59	18.6	18.4	40.28	50.43	6	12	1	1	0.64
183	11.81	16.3	54.1	4.97	16.6	22.3	44.06	48.39	6	11	1	6	0.58
190	12.31	16.8	54.7	5.63	18.0	23.3	41.72	44.18	9	2½	1	13	0.54
209	14.69	18.0	60.9	6.77	17.5	28.9	39.26	44.53	6	11¼	1	6	0.54

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TABLE G Continued — NORMAL PREGNANCY - 42 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight		Placenta weight		Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb.	ozs	lb.	ozs	
214	13.31	16.6	59.8	6.16	16.6	27.7	42.86	49.89	8	15	1	7	0.54
223	13.35	18.0	55.4	3.57	18.1	14.7	45.56	51.58	8	1	1	11½	0.73
227	13.24	18.6	53.1	5.74	18.4	23.3	39.81	45.84	9	3	2	0¾	0.57
228	12.60	17.2	54.7	5.78	17.2	25.1	39.96	46.81	6	11	1	4	0.54
232	10.71	17.5	45.7	3.93	17.2	17.1	41.74	45.84	6	9½	1	7¾	0.63
234	11.14	18.4	45.2	2.58	18.5	10.4	47.37	52.82	7	7	1	9	0.77
AVERAGE (19)	12.60	17.7	53.5	4.86	17.7	20.5	41.55	48.10	7	13	1	8	0.62
RANGE	9.59	13.4	37.3	1.84	12.6	10.4	34.89	44.18	6	4	1	1	0.44
	15.55	21.0	64.9	7.89	22.2	35.3	47.37	52.82	9	6	2	5	0.81

Gross infarction of Placenta:- Cases 90; 153 only

No cases induced surgically or by Pitocin Drip

TABLE H

NORMAL PREGNANCY - 43 WEEKS

Case No.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight		Placenta weight		Coefft. of Oxygen Utiln.
	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb.	ozs	lb.	ozs	
1	15.16	22.0	51.4	6.82	22.5	22.6	42.12	47.77	7	6¼	1	3	0.55
3	13.36	18.2	54.8	5.24	18.5	21.1	43.91	48.98	7	5½	1	4	0.61
22	14.34	17.0	62.9	4.37	17.1	19.1	43.47	51.60	6	8½	1	7	0.70
110	12.84	17.4	55.1	3.58	17.0	15.7	42.06	47.37	7	11¾	1	5	0.72
165	12.57	18.4	51.0	3.89	18.4	15.8	38.21	45.66	7	5¼	1	1	0.69
215	12.01	16.3	55.0	5.43	16.6	24.4	44.76	49.06	8	8	1	7	0.55
229	13.04	17.8	54.7	3.97	18.0	16.5	40.41	46.20	7	7½	1	7	0.70
AVERAGE (7)	13.33	18.2	55.0	4.76	18.3	19.3	42.13	48.09	7	7½	1	5	0.65
RANGE	12.01	16.3	51.0	3.58	16.6	15.7	38.21	45.66	6	8½	1	1	0.55
	15.16	22.0	62.9	6.82	22.5	24.4	44.76	51.60	8	8	1	7	0.72

Gross infarction of Placenta:- Cases No. 1; 22 only

Pitocin Drip Induction:- Cases 165, 215

TABLE I

CORD OBSTRUCTION AND DELAYED DELIVERY

Case No.	Durn. of Pregny.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight lb. ozs	Remarks
		Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.		
CORD ROUND NECK											
192	36	11.93	19.3	46.1	3.88	19.3	15.0	38.25	42.66	4 11	Foetal distress Baby died Intraventricular Haemge
76	39	11.10	15.0	55.2	2.56	15.5	12.3	45.11	54.57	7 9	
166	40	4.11	14.9	20.6	2.14	14.3	11.2	42.24	47.24	8 11¼	Cord twice round neck Baby limp
11	40	16.78	18.4	68.1	2.20	17.6	9.3	41.15	53.04	8 7½	
51	40	12.18	17.5	51.9	2.94	17.5	12.5	46.31	50.76	7 0¾	
78	41	15.44	17.4	66.2	2.13	18.8	8.5	44.69	51.58	8 5	Cardiac IIB Oxygen given in labour
100	41	20.35	20.3	74.8	4.09	18.3	16.7	37.67	49.74	8 10	Baby very limp
AVERAGE (6)		13.33	17.3	56.1	2.68	17.0	11.7	42.86	51.15	8 2	
RANGE/											

TABLE I Continued — CORD OBSTRUCTION AND DELAYED DELIVERY

Case No.	Durn. of Pregny.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight	Remarks
		Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.	lb. ozs	
RANGE		4.11	14.9	20.6	2.13	14.3	8.5	37.67	47.24	7 0¾	
		20.35	20.3	74.8	4.09	18.8	16.7	46.31	54.57	8 11¼	
DELAYED DELIVERY											
42	40	13.71	18.6	55.0	4.72	18.7	18.8	42.22	47.33	6 3¾	Difficulty with shoulders
31	40	14.97	19.6	57.0	4.07	18.9	16.1	35.03	48.26	7 10¼	"
204	40	8.33	18.5	33.6	2.36	18.6	9.5	40.61	50.24	6 5¾	Head on perineum 30 mins.
AVERAGE (3)		12.34	18.9	48.5	3.72	18.7	14.8	39.29	48.61	6 12	

TABLE J

TWIN PREGNANCY

Case No.	Durn. of Pregny.	Umbilical Vein			Umbilical Arteries			CO_2 vols.%		Baby weight lb. ozs	Placenta weight lb. ozs	Remarks
		Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Oxygen Vols.%	Hb. Gms.%	Oxygen Satn.%	Umb. Vein	Umb. Arts.			
14	32	6.89	14.0	36.7	3.91	14.6	20.00	45.30	49.52	3 14½)	1 14	Vertex
		8.43	15.1	41.7	3.96	15.3	19.3	44.14	48.45	3 12½)		Breech (Binovular)
114	34	12.89	17.7	54.4	8.20	17.2	35.6	43.07	46.62	3 11)	2 8	Vertex
115		11.11	20.0	41.5	6.09	19.8	23.0	42.30	47.13	4 4¼)		Breech (Binovular)
158	36	6.31	18.3	25.7	2.20	18.0	9.1	41.38	48.20	4 2¾)	2 3	Vertex
		7.28	17.8	30.5	3.08	19.3	11.9	44.28	47.98	5 8¾)		Breech (Binovular)
117	38	10.78	17.1	47.1	5.12	17.3	22.1	34.27	38.25	5 1½		Vertex
AVERAGE (7)		9.10	17.1	39.7	4.65	17.4	20.1	42.11	46.59			
RANGE		(6.31	14.0	25.7	2.20	14.6	9.1	34.27	38.25			
		(12.89	20.0	54.4	8.20	19.8	35.6	45.30	49.52			

TABLE K — PRE-ECLAMPSIA — SPONTANEOUS VERTEX DELIVERIES - CHLOROFORM - (39 - 41 WEEKS)

Case No.	Durn. of Preg.	Umbilical Vein			Umbilical Arteries			A-V Diffce %	CO_2 vols.%		Baby Wt. lb.oz.	Plac[a] Wt. lb.oz.	Gross Infn	Coefft of Oxygen Utiln.	B.P. at Dely	Max[m] B.P.	Alb[a] Gm/ Litre	Oed[a]	Dur[n] of Tox[a]	Admission to Hospital	Booked or not	Induction
		Oxygen vols.%	Hb Gms.%	Oxygen Satn.%	Oxygen vols.%	Hb Gms.%	Oxygen Satn.%		Umb. Vein	Umb. Arts.												
GROUP 1.	GENERALISED OEDEMA AND EXCESSIVE WEIGHT GAIN																					
66	39	12.31	18.6	49.4	4.01	18.1	16.5	32.9	43.04	49.94	7 12¾	1 10	-	0.67	136/88	140/90	-	++	2/12	34-37 weeks	B	Readmitted in labour
147	40	12.20	16.0	56.9	3.88	16.3	17.8	39.1	47.62	52.08	7 6	1 7	+	0.68	120/80	134/82	-	+++	2/12	39 weeks	B	Nil
167	40	11.58	14.6	59.2	4.64	13.9	24.9	34.3	39.85	48.34	7 14	1 11	-	0.60	120/86	140/90	In labour +(x1)	++	2/52	38 weeks	B	Readmitted in labour
56	41	13.25	19.2	51.5	6.47	19.4	24.9	26.6	38.90	43.91	6 15½	1 6	-	0.51	126/84	130/90	-	++	2/12		B	Nil
AVERAGE (4)		12.34	17.1	54.3	4.75	16.9	21.0	33.3	42.35	48.57	7 8	1 8½		0.62								
GROUP 2.	MILD PRE-ECLAMPSIA																					
188	39	11.89	17.6	50.4	3.38	17.9	14.1	36.3	41.73	47.59	7 15¾	1 10	-	0.72	130/90	146/108	-	+	1/12	39 weeks	B	Nil
91	40	13.11	16.9	57.9	4.82	17.5	20.6	37.3	39.99	45.79	8 15	1 7	-	0.63	150/100	150/90	In labour ++	++	1/12		B	Admitted in labour
136	41	13.18	17.8	55.3	3.57	18.2	14.6	40.7	44.38	50.45	7 12	1 9	-	0.73	140/90	140/90	+	Trace	1/52		B	Admitted in labour
152	41	12.39	17.4	53.1	5.12	17.3	22.1	31.0	36.96	42.05	7 9½	1 3	-	0.59	140/85	160/80	0.5	Trace	2/52	40 weeks	B	Nil
213	41	12.29	18.9	48.5	2.73	18.8	10.8	37.7	45.95	49.45	7 12½	1 4½	+	0.78	134/76	150/100	In labour ++	++	1/12	40 weeks	N.B.	Pitocin drip
AVERAGE (5)		12.57	17.7	53.0	3.92	17.9	16.4	36.6	41.80	47.07	8 0	1 6¾		0.69								
GROUP 3.	MODERATE PRE-ECLAMPSIA																					
118	39	14.15	19.6	53.9	5.77	19.4	22.2	31.7	45.72	53.68	7 15	1 7	-	0.58	146/104	150/96	+	++	5/52	35-36 weeks	B	Readmitted in labour
176	39	12.73	18.1	52.5	4.94	17.7	20.8	31.7	43.96	52.34	6 2	1 6	-	0.61	164/110	170/100	+	++	<3/52	38 weeks	B	Nil
17	40	13.72	18.7	54.8	6.53	18.9	25.8	29.0	35.92	42.70	8 15½	2 0	-	0.52	164/100	150/90	1.0	++	1/12	39 weeks	B	Artl. rupture of Membranes
96	40	7.53	17.1	47.5	3.55	16.9	15.7	31.8	47.53	53.74	5 15¾	1 3	++	0.53	145/100	190/110	0.25	++	?3/52	38+ weeks	N.B.	Nil

TABLE K Continued — PRE-ECLAMPSIA — SPONTANEOUS VERTEX DELIVERIES - CHLOROFORM - (39 - 41 WEEKS)

Case No.	Durn. of Preg.	Umbilical Vein			Umbilical Arteries			A-V Diffce %	CO_2 vols.%		Baby Wt.	Plac^{a} Wt.	Gross Inf^{n}	Coefft of Oxygen Utiln.	B.P. at Del^{y}	Max^{m} B.P.	Alb^{a} Gm/ Litre	Oed^{a}	Durn. of Tox^{a}	Admission to Hospital	Booked or not	Induction
		Oxygen vols.%	Hb Gms.%	Oxygen Satn.%	Oxygen vols.%	Hb Gms.%	Oxygen Satn.%		Umb. Vein	Umb. Arts.	lb.oz.	lb.oz.									
GROUP 3 Continued																					
162	40	10.96	15.6	52.4	4.96	15.5	23.9	28.5	42.62	48.20	7 5½	1 9	-	0.55	146/98	180/115	+	+	3/52	39 weeks	B	Artl. Rupture of Membranes
197	40	7.87	19.9	29.5	1.79	19.6	6.8	22.7	46.55	49.29	8 2½	2 4	+	0.77	160/110	150/100	+	++	7/52	33-34 weeks	B	Medical
217	40	12.67	16.9	56.0	5.96	17.1	26.0	30.0	45.73	49.42	8 4	1 8½	-	0.53	130/90	170/90	Trace	++	1/12	36 weeks	B	Nil
89	41	12.68	18.3	51.7	3.72	18.2	15.3	36.4	46.81	52.51	6 13½	1 8	-	0.75	150/100	190/105	3.0	++	7/52	35 weeks	N.B.	Nil
121	41	15.38	19.4	59.2	4.28	19.0	16.8	42.4	41.04	52.01	8 7	1 6	-	0.72	116/88	140/100	0.75	+	?2/52	40 weeks	B	Artl. Rupture of Membranes
151	41	12.34	17.5	52.6	5.23	18.2	21.4	31.2	41.54	47.95	7 11½	1 4	-	0.58	178/110	168/88	++	++	3/52		B	Admitted in labour
AVERAGE(10)		12.00	18.1	51.0	4.67	18.1	19.5	31.5	43.74	50.18	7 9¼	1 8¾		0.61								
GROUP 4. SEVERE PRE-ECLAMPSIA																						
191	39	6.54	18.2	26.8	1.66	17.9	6.9	19.9	44.45	49.06	4 0	0 15	+	0.75	160/105	185/126	18.0	++	6/52	34 weeks	N.B.	Artl. Rupture of Membranes Pitocin drip
225	39	12.07	17.6	51.2	5.45	17.7	23.0	28.2	43.46	48.56	9 3	1 11	-	0.55	210/140	154/104	++	+	?1/12		N.B.	Admitted in labour
131	40	8.97	19.9	33.6	3.55	19.7	13.5	20.1	38.36	44.05	5 10½	1 0	+	0.60	152/128	160/115	0.25	++	5/52	35+ weeks	N.B.	Pitocin drip
130	41	14.29	17.9	59.6	5.90	17.9	24.6	35.0	34.64	42.42	6 14¾	1 6	-	0.59	170/130	170/130	2.0	+++	?1-2/52	1 day before delivery Post partum fit	B	Artl. Rupture of Membranes
AVERAGE (4)		10.47	18.4	42.8	4.14	18.3	17.0	25.8	40.23	46.02	6 7	1 4		0.62								
TOTAL AVERAGE(23)		11.92 ±2.21	17.9 ±1.35	50.6 ±8.89	4.43	17.9	18.7 ±5.79	31.9	42.47 ±3.85	48.50	7 7½	1 7½		0.63								

TABLE L

PRE-ECLAMPSIA PREMATURE AND POSTMATURE DELIVERIES (SPONTANEOUS)

Case No.	Durn. of Preg.	Umbilical Vein			Umbilical Arteries			A-V Diffce %	CO_2 vols.%		Baby Wt.	Plac[a] Wt.	Coefft of Oxygen Utiln.	Gross Infn	B.P. at Dely	Max[m] B.P.	Alb[a] Gm/ Litre	Oed[a]	Durn of Tox[a]	Admission to Hospital	B'ked or not	Class[n] Tox[a]	Induction
		Oxygen vols.%	Hb Gms.%	Oxygen Satn.%	Oxygen vols.%	Hb Gms.%	Oxygen Satn.%		Umb. Vein	Umb. Arts.	lb. oz.	lb. oz.											
5	33	12.99	15.9	61.0	4.23	16.5	19.1	41.9	40.81	45.80	3 1½	0 12	0.67	+	170/100	206/100	2.0	+	1/12	BP 170/100 at 11 weeks	B	Severe on Ess[L] Hyp[n] Cord round neck	A.R.M. and P.Drip
87	33	12.65	18.0	52.4	3.79	18.8	15.0	37.4	47.98	56.49	3 3¼	0 12	0.70	+	160/110	190/128	3.75	+++	<1/12	32 weeks	B	Severe	A.R.M.
79	35	8.53	16.4	38.8	3.70	16.3	16.9	21.9	36.98	43.18	4 0	1 1	0.57	+	170/105	185/115	8.5	++	?3/52	34 weeks	N.B.	Severe	A.R.M. & P.Drip
178	35	9.52	19.4	36.6	4.42	18.9	17.5	19.1	41.36	45.51	5 8	1 5	0.54	+	160/110	200/130	1.3	+++	5/52	31+weeks	B	Severe	A.R.M.
67	36	13.42	19.0	52.7	6.19	19.4	23.8	28.9	45.41	49.58	5 1	1 1½	0.54	+	165/105	152/104	+++	++	?	35+weeks	N.B.	Severe	A.R.M.
53	38	11.93	12.8	69.6	4.85	12.7	28.5	41.1	42.58	48.37	7 6	1 7	0.59	Eryth	146/100	150/90	+	++	2/52	Rh.Neg. 2 days before	B	Mild	A.R.M.
70	38	12.33	17.7	52.0	5.11	18.4	20.7	31.3	40.19	45.55	6 1¾	1 2	0.59	-	185/125	185/125	0.9	++	?1/12	37 weeks	N.B.	Severe	Nil
75	38	12.74	18.3	52.0	5.92	17.7	25.0	27.0	44.19	49.01	7 4¼	1 7	0.54	-	150/100	155/105	8.0	++	?1/12	1 day before	N.B.	Mod.	Nil
182	38	13.24	19.9	49.7	5.02	20.0	18.7	31.0	37.52	47.75	7 6½	1 6	0.62	-	140/90	170/100	0.5	++	2/12	35+weeks	B	Mod.	Nil
220	38	13.16	17.1	57.4	5.84	17.4	25.1	32.3	42.36	47.61	6 15	1 7½	0.56	-	146/100	152/100	+	Trace	<1/12	37 weeks	B	Mod.	P.Drip A.R.M.
AVERAGE(9)		11.95 ±1.74	17.6 ±2.1	51.2 ±9.69	4.98	17.7	21.2 ±4.57	30.0	42.06 ±3.56	48.12	5 14	1 3¾	0.58										
RANGE		8.53	12.8	36.6	3.70	12.7	15.0		36.98	43.18	3 3¼	0 12	0.54										
		13.42	19.9	69.6	6.19	20.0	28.5		47.98	56.49	7 6½	1 7½	0.70										
62	42	8.36	17.8	35.1	3.22	17.8	13.5	21.6	39.78	43.65	8 15½	1 12	0.61	+	145/100	150/100	-	++	1/12	39+weeks	B	Mild	Medical
154	42	13.74	18.2	56.3	2.01	18.1	8.3	48.0	39.85	45.26	9 4	1 12	0.85	-	180/115	160/105	+	+	2/52+	40+weeks	N.B.	Mod.	Medical
155	42	12.32	16.9	54.4	1.41	17.1	6.2	48.2	40.49	46.41	8 9	1 9	0.89	+	160/100	180/100	+	Trace	2/52+	1 day before	N.B.	Mod.	A.R.M.

TABLE L Continued — PRE-ECLAMPSIA PREMATURE AND POSTMATURE DELIVERIES (SPONTANEOUS)

Case No.	Durn. of Preg.	Umbilical Vein			Umbilical Arteries			A-V Diffce %	CO_2 vols.%		Baby Wt. lb. oz.	Plac[a] Wt. lb. oz.	Coefft of Oxygen Utiln.	Gross Inf[n]	B.P. at Dely	Max[m] B.P.	Alb[a] Gm/ Litre	Oed[a]	Dur[n] of Tox[a]	Admission to Hospital	B'ked or not	Class[n] Tox[a]	Induction
		Oxygen vols.%	Hb Gms.%	Oxygen Satn.%	Oxygen vols.%	Hb Gms.%	Oxygen Satn.%		Umb. Vein	Umb. Arts.												
134	42	12.08	21.0	42.9	5.84	20.3	21.5	21.4	44.29	48.71	9 $4\frac{3}{4}$	1 11	0.52	+	$\frac{146}{96}$	$\frac{140}{100}$	Trace	+++	$\frac{1}{12}$	40+weeks	B	Mild	Nil
141	42	8.09	15.7	38.5	3.92	16.1	18.2	20.3	40.08	45.82	8 $10\frac{1}{2}$	1 14	0.52	++	$\frac{170}{110}$	$\frac{150}{88}$	In labour ++	Trace	$\frac{3}{52}$	In labour	B	Mild	Nil
138	?44	11.73	20.5	42.7	5.41	20.4	19.8	22.9	42.19	50.28	7 $15\frac{3}{4}$	1 7	0.54	+	$\frac{120}{80}$	$\frac{130}{80}$	+	++	$>\frac{1}{12}$	?41 weeks	B	Mild	
AVERAGE(6)		11.05	18.4	45.0	3.64	18.3	14.6	30.4	41.11	46.69	8 $12\frac{1}{2}$	1 $10\frac{3}{4}$	0.66										
RANGE		8.09	15.7	35.1	1.41	16.1	6.2		39.78	43.65	7 $15\frac{3}{4}$	1 7	0.52										
		13.74	21.0	56.3	5.84	20.4	21.5		44.29	50.28	9 $4\frac{3}{4}$	1 14	0.89										

TABLE M

PREGNANCY WITH HYPERTENSION SPONTANEOUS VERTEX DELIVERIES - CHLOROFORM

Case No.	Durn. of Preg.	Umbilical Vein			Umbilical Arteries			A-V Diffce %	CO_2 vols.%		Baby Wt. lb. oz.	Plac[a] Wt. lb. oz.	Gross Infn	Coefft of Oxygen Utiln.	B.P. at Dely	Max[m] B.P.	B.P. early Preg.	Alb[a]	Oed[a]	Admission	Remarks
		Oxygen vols.%	Hb Gms.%	Oxygen Satn.%	Oxygen vols.%	Hb Gms.%	Oxygen Satn.%		Umb. Vein	Umb. Arts.											
8	37	18.51	17.1	80.8	10.63	17.3	45.9	34.9	42.55	46.95	6 8¼	1 5	-	0.43	160/90	170/98	180/80	-	-		
185	39	15.92	17.3	68.7	8.94	18.4	36.3	32.4	41.35	43.67	7 9	1 13	-	0.44	150/90	170/100	140/70	-	-	34-36 and then for induction	Artl. rupture of membranes
222	39	17.15	17.7	72.3	9.12	17.9	38.0	34.3	41.18	45.22	7 15	1 8½	-	0.47	140/100	160/92	140/90	-	-	32-33: 37+ →	Hypertension prev. preg.
52	40	17.90	19.6	68.1	10.05	19.8	37.9	30.2	37.10	43.97	6 11½	1 3	-	0.44	150/105	160/100	130/80	-	-	In labour	Hypertension prev. preg.
111	40	16.44	17.8	68.9	6.64	17.2	28.8	40.1	43.33	50.10	6 12¼	1 4	-	0.70	140/84	180/100	140/100	-	-	34+	
120	40	18.61	18.6	74.7	9.43	18.0	39.1	35.6	40.41	47.40	5 13¼	1 4	+	0.49	140/90	145/95	140/80	-	-	In labour	
216	40	15.30	15.8	72.3	7.52	15.6	36.0	36.3	44.14	47.97	7 12	1 12½	-	0.51	150/96	166/100	166/100	-	-	22: 3 days before delivery	
93	40	18.92	18.4	76.7	7.17	17.9	29.9	46.8	41.20	48.91	8 11¾	1 13	+	0.62	140/105	160/85	?	-	-	36+	
235	41	16.60	18.4	67.3	8.17	18.8	32.4	34.9	44.69	48.19	7 4	1 7	-	0.51	140/90	140/102	150/88	-	-	In labour	
AVERAGE(8)		17.11 ±1.29	18.0 ±1.1	71.1 ±3.41	8.38	18.0	34.8 ±3.92	36.3	41.68 ±2.42	46.93	7 5	1 8		0.52							
RANGE		15.30	15.8	67.3	6.64	15.6	28.8		37.10	43.67	5 13¼	1 3		0.44							
		18.92	19.6	76.7	10.05	19.8	39.1		44.69	50.10	8 11¾	1 13		0.70							

TABLE N

FORCEPS DELIVERIES

Case No.	Durn. of Preg.	Umbilical Vein			Umbilical Arteries			A - V Diffce %	CO_2 content		Baby Wt.	Baby Condⁿ	B.P.	Albᵃ Gm/ Litre	Oedᵃ	Induction	Anaesthetic	Indication for Forceps
		Oxygen vols.%	Hb Gms.%	Oxygen Satn.%	Oxygen vols.%	Hb Gms.%	Oxygen Satn.%		Umb. Vein	Umb. Arts.	lb. oz.							
NORMAL PREGNANCY																		
21	40	7.62	18.3	31.3	2.16	18.0	9.0	22.3	39.34	44.22	7 2½	F.G.					Flax:Cyclo:N_2O oxygen	Foetal distress Fibroids
27	40	11.46	17.9	47.8	5.49	17.5	23.4	24.4	43.02	48.05	7 9½	Limp					Oxygen:Cyclo & oxygen	Foetal distress Cardiac IIA
33	40	19.19	17.5	81.8	——	——	——	——	40.11	——	8 1	F.G. (Cord round neck)					Flax:Cyclo: oxygen	Second stage delay Inertia
35	41	18.51	18.4	75.1	2.91	18.7	11.6	63.5	38.16	47.82	9 4½	Fair					Flax:Cyclo: oxygen	Foetal distress
43	41	11.84	20.5	43.1	5.16	20.1	19.2	23.9	44.34	47.31	6 14¼	F.G.					Flax:Cyclo: oxygen	Deep transverse arrest
AVERAGE (5)		13.72	18.5	55.8	3.93	18.6	15.8	40.0	40.99	46.85	7 12¾							
HYPERTENSION																		
50	41	16.15	18.0	67.0	7.92	17.9	33.0	34.0	48.95	54.30	8 0¾	F.G.	180/100	-	-		Flax:Cyclo: oxygen	Second stage delay Inertia
PRE-ECLAMPSIA																		
16	37	2.85	16.7	12.7	1.88	16.5	8.5	4.2	41.24	44.03	5 10¾	F.G.	170/110	1.75	Trace	-	Flax:N_2O:ether: oxygen	Second stage delay
113	39	11.37	20.2	42.0	3.86	21.0	13.7	28.3	48.45	55.28	7 8	Good	170/110	3.0	+	A.R.M.	Flax:cyclo: oxygen	Deep transverse arrest
10	39	9.67	17.6	41.0	2.54	17.6	10.8	30.2	47.08	52.09	7 15½	F.G.	170/90	0.5	+	A.R.M.	N_2O:cyclo: Oxygen	Foetal distress
37	39	6.83	16.2	31.5	3.16	16.2	14.6	16.9	44.86	50.28	8 12¾	F.G.	160/120	++	+	-	Pent:cyclo: oxygen	Maternal distress
48	39	11.43	13.9	61.4	——	——	——	——	48.57	——	7 3	Limp	155/100	Trace	+	-	Flax:cyclo: oxygen	Persistent occipito posterior
15	40	12.41	19.3	48.0	4.37	19.4	16.8	31.2	44.04	50.28	7 8	Limp	160/110	++	++	-	Flax:cyclo:N_2O: oxygen	Second stage delay
58	40	9.04	17.8	37.9	2.04	18.0	8.5	29.4	50.43	55.24	7 6¼	F.G.	150/95	-	++	-	Flax:cyclo: oxygen	Second stage delay Inertia
25	41	12.38	18.3	50.5	5.08	18.2	20.8	29.7	45.93	53.12	10 2	F.G.	180/120	++	++	-	Trilene:N_2O: oxygen	Fulminating toxaemia
64	42	17.76	18.8	70.5	8.08	18.9	31.9	38.6	45.18	50.43	9 4	Good	150/100	Trace	++	-	Flax:cyclo: oxygen	Maternal distress
AVERAGE (9)		10.42	17.6	43.9	3.88	18.2	15.7	28.2	46.20	51.34	7 15							

TABLE O

NORMAL PREGNANCY CAESAREAN SECTION DELIVERIES

Case No.	Durn. of Preg.	Umbilical Vein			Umbilical Arteries			A-V Diffce %	CO_2 vols.%		Baby Wt.	Plac[a] Wt.	Indication for Section	Anaesthetic	Remarks
		Oxygen vols.%	Hb Gms.%	Oxygen Satn.%	Oxygen vols.%	Hb Gms.%	Oxygen Satn.%		Umb. Vein	Umb. Arts	lb. oz.	lb. oz.			
ELECTIVE															
A. LOWER SEGMENT SECTION															
205	39	6.40	16.4	29.1	2.00	16.1	9.3	19.8	56.08	58.35	8 0	1 3	Cardiac cont[d] pelvis. Previous C/S	Flax:cyclo: oxygen	Baby slow to respond
69	39	8.71	16.0	40.6	3.07	16.0	14.3	26.3	54.73	57.95	9 10¼	1 5	Para 2 (dead). Breech	Flax:Pent: N_2O:cyclo:O_2	
175	39	15.62	18.2	64.1	7.73	18.2	31.7	32.4	43.24	53.13	5 9¾	0 15	Prev. C/S. Plac. Praevia IV	Flax:cyclo: oxygen	
184	39	6.44	16.1	29.9	1.93	16.9	8.5	21.4	42.64	47.77	6 15	1 1	Para 1 (dead) Breech 37 yrs.	Flax:cyclo: oxygen	Baby died ($\frac{5}{52}$) Indeterminate sex
238	39	5.06	17.8	21.2	2.00	18.0	8.3	12.9	51.51	56.97	6 14¾	1 0½	Prev. C/S. Transverse lie	Flax:cyclo: oxygen	Difficult delivery Baby limp
102	40	7.03	16.5	31.8	3.62	16.8	16.1	15.7	56.40	57.63	7 12½	1 13	Prev. C/S. Cont[d] pelvis	Flax:Pent:N_2O: cyclo:O_2	
109	40	6.07	14.0	32.4	1.62	14.2	8.5	23.9	52.49	55.44	6 10¼	1 5	Prev. C/S. Cont[d] pelvis	Flax:Pent:N_2O: cyclo:O_2	Cord loosely round neck
AVERAGE (7)		7.90	16.4	35.6	3.14	16.6	13.8	21.8	51.01	55.32	7 5¾	1 3¾			
237	42	7.35	16.3	33.7	2.66	16.3	12.2	21.5	51.03	55.19	7 4½	1 3	Contracted pelvis. 35 yrs.	Flax:Pent:N_2O: oxygen	Bleeding ++ before delivery
189	44	8.46	16.9	37.4	1.95	16.9	8.6	28.8	41.43	48.65	9 9¾	1 10	Post maturity. Pit drip failed	Local	No liquor. Yellow staining ++
B. CLASSICAL SECTION															
23	39	12.46	16.1	57.8	7.00	16.4	31.9	25.9	44.02	47.98	8 1½	1 10	Prev. C/S. Fibroids. Cardiac	Flax:Pent: cyclo:oxygen	
IN LABOUR (LOWER SEGMENT)															
30	38	13.45	14.7	68.3	10.29	15.5	49.5	18.8	53.26	56.41	7 1½	1 3	Inertia. Prev. myomectomy	Flax:Pent: cyclo:oxygen	
88	40	14.72	14.9	73.7	8.68	15.6	41.5	32.2	44.46	51.71	8 14¾	1 13	Cervical dystocia Maternal distress	Spinal:oxygen	Prolonged labour (80 hrs.)
163	41	8.27	17.5	35.3	1.71	17.2	7.4	29.7	44.40	47.96	7 13½	1 5	Cont[d] pelvis. Trial of labour	Flax:cyclo: oxygen	Baby very shocked
AVERAGE (3)		12.15	15.7	59.1	6.89	16.1	32.8	26.3	47.37	52.03	7 15¼	1 7			
TOTAL AVERAGE (13)		9.23	16.3	42.7	4.17	16.5	19.1	23.6	48.90	53.47	7 11½	1 5½			

TABLE P

TOXAEMIC PREGNANCY CAESAREAN SECTION DELIVERIES

Case No.	Durn. of Preg.	Umbilical Vein			Umbilical Arteries			A - V Diffce %	CO_2 vols.%		Baby Wt. lb. oz.	Plac[a] Wt. lb. oz.	Baby Cond[n]	B.P.	Alb[a] Gm/ Litre	Oed[a]	Indication for Section	Anaesthetic	Remarks
		Oxygen vols.%	Hb Gms.%	Oxygen Satn.%	Oxygen vols.%	Hb Gms.%	Oxygen Satn.%		Umb. Vein	Umb. Arts.									
CLASSICAL SECTION																			
18	35	12.83	18.9	50.7	3.98	19.3	15.4	35.3	49.24	57.76	3 8½	0.13	Fair	236/128	8.5	+++	Fulminating Toxaemia	Flax:cyclo: oxygen	Baby died. intra-pulmonary haemorrhage
LOWER SEGMENT SECTION																			
A. NOT IN LABOUR																			
108	32	9.04	17.9	37.7	3.44	18.2	14.1	23.6	50.97	56.82	4 4	1 3	Fair	170/110	16.0	+++	Severe Toxaemia	Spinal	Baby died. Pre-maturity. Hyaline membrane
187	32	6.26	19.9	23.5	1.79	20.1	6.7	16.8	43.93	50.18	3. 0	0 11	Fair	140/110	12.0	++	Eclampsia (1 fit)	Spinal	Normal 3/52 before admission 1 fit at home.
198	36	12.72	18.8	50.5	6.16	18.8	24.5	26.0	45.66	50.26	5 9	1 2	F.G.	180/108	1.0	+++	Previous C/S	Spinal	Moderate toxaemia
173	38	9.87	15.7	46.9	3.00	15.7	14.3	32.6	51.13	54.71	8 0½	1 0	F.G.	170/90	0.75	+	Cardiac. Plac. Praevia III	Flax:Pent: cyclo:O_2	
AVERAGE (4)				39.7			14.9	24.8											
B. IN LABOUR																			
196	38	8.70	19.5	33.3	1.28	19.5	4.9	28.4	41.98	48.76	6 9	1 5	Fair	146/112	++	++	Foetal distress Brow	Flax:Pent: cyclo:N_2O:O_2	30 hours in labour
168	40	4.33	17.2	18.8	1.48	17.2	6.4	12.4	53.39	54.05	6 13	1 5	V. limp	164/104	3.0	+	Foetal distress	Flax:Pent: ether:cyclo: O_2	Severe Toxaemia A.R.M.
180	41	12.32	15.7	58.6	5.64	16.6	25.4	33.2	44.84	49.41	9 5½	1 9	Good	148/104	In labour +	+	Inertia	Cyclo:ether: oxygen	Mild Toxaemia
24	42	3.40	18.3	13.9	1.07	18.5	4.3	9.6	55.39	59.85	9 2½	1 7	Limp	140/90	Trace	Trace	Inertia	Flax:Pent: cyclo:N_2O:O_2	Mild Toxaemia 57 hours in labour
137	42	4.96	14.2	26.1	1.70	13.9	9.1	17.0	41.94	44.56	8 2	2 0	Limp	160/95	++	-	Contracted pelvis Failed trial. Brow.	Flax:cyclo: oxygen	Mild Toxaemia
C. HYPERTENSION																			
72	39	5.01	16.9	22.1	2.76	17.2	12.0	10.1	52.40	56.68	9 1	1 7	Fair	145/100	-	Trace	Previous C/S.	Flax:cyclo: oxygen	Mild hypertension Cord round baby Elective.
D. CHRONIC NEPHRITIS																			
193	39	4.77	15.1	23.6	2.06	15.3	10.1	13.5	47.92	52.49	7 8	1 4	Fair	160/105	1.25	Trace	2 prev. C/S.	Cyclo:N_2O: oxygen	Elective

TABLE Q

DIABETES MELLITUS CAESAREAN SECTION

Case No.	Durn. of Preg.	Umbilical Vein			Umbilical Arteries			A-V Diffce %	CO_2 vols.%		Baby Wt.	Plac^a Wt.	Cond. of Baby	Indication for Section	Anaesthetic	Remarks
		Oxygen vols.%	Hb Gms.%	Oxygen Satn.%	Oxygen vols.%	Hb Gms.%	Oxygen Satn.%		Umb. Vein	Umb. Arts.	lb. oz.	lb. oz.				
LOWER SEGMENT SECTION														**Diabetes Mellitus in each plus**		
A. NO ADDITIONAL COMPLICATION																
179	35	5.83	15.2	28.6 ✓	0.54	14.6	2.8	25.8	44.07	51.84	9 15	2 2	Fair	Previous C/Section	Spinal	Hydramnios + Baby died. Renal thrombosis
242	35	11.11	17.3	47.9 ✓	5.57	17.4	23.9	24.0	49.54	55.72	8 15	1 6			Spinal	Hydramnios
169	36	12.26	15.2	60.2 ✓	4.73	14.4	24.5	35.7	48.61	59.59	6 13½	1 7	F.G.		Spinal+oxygen	
200	?36	3.69	16.8	16.4 ✓	0.77	15.7	3.7	12.7	44.50	50.85	6 6¼	1 6	F.G.	Previous C/Section	Spinal	Mongol
226	36	3.11	17.1	13.6 ✓	1.02	17.0	4.5	9.1	54.48	57.56	7 7¾	1 3			Spinal	Cord twice round neck
236	36	2.46	16.7	11.0 ✓	0.81	16.9	3.6	7.4	52.28	54.13	9 0	1 8			Spinal+oxygen	Delay Uterine contraction
195	38	13.06	18.4	53.0 ✓	5.50	18.6	22.1	30.9	47.43	54.35	6 10½	1 13	F.G.		Spinal	Pitocin drip failed
AVERAGE (7)		7.36	16.7	33.0	2.71	16.4	12.2	20.8	48.70	54.86	7 14¼	1 8¾				
B. TOXAEMIA AND DIABETES MELLITUS																
170	36	2.64	17.9	11.0 ✓	0.81	17.2	3.5	7.5	48.45	50.15	5 13	1 1	Fair		Spinal	
219	36	7.69	18.5	31.0 ✓	0.62	18.8	2.5	28.5	49.73	54.80	6 10	1 1½			Spinal	Delay Uterine contraction
61	36	10.46	17.5	44.6 ✓	1.49	17.8	6.3	38.3	47.84	56.82	8 10	1 6			Spinal	
AVERAGE (3)		6.93	18.0	28.9	0.97	17.9	4.1	24.8	48.67	53.92	7 0¼	1 2¾				
CLASSICAL SECTION																
63	35	13.04	13.6	71.6 ✓	8.47	13.8	45.8	25.8	48.42	53.72	5 6¼	1 0		Previous C/Section	Flax:Pent: cyclo:N_2O:O_2	Diabetic Nephropathy
174	36	7.20	15.7	34.2 ✓	0.81	15.5	3.9	30.3	55.84	61.97	8 6	1 12	F.G.	Previous C/Section	Flax:Pent: cyclo:N_2O:O_2	
171	37	14.11	17.6	59.8 ✓	4.41	17.7	18.9	41.2	48.57	57.80	7 6½	1 0½	F.G.	Previous C/Section	Spinal	
AVERAGE (3)		11.45	15.6	55.2	4.56	15.7	22.8	32.4	50.94	57.83	7 0¾	1 4¼				
TOTAL AVERAGE (13)		8.20	16.7	37.1	2.73	16.6	12.7	24.4	49.21	55.33	7 8	1 6¼				